A TREATISE
ON
TROPICAL SKIN DISEASES

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BY

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FOREWARD

By

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Dr Kali Das Lahiri, M B, B S (Patna), M. D. (Dermatology) Zurich, Ph D (Dermatology) Edinburgh, D T M & H (London), Assistant Professor of Dermatology in the Calcutta Medical College has written this book "Treatise on Tropical Skin Diseases" With his twenty years experience as a clinician, teacher and research worker in tropical skin diseases, Dr Lahiri, is eminently qualified to write such a book

I have read the book with interest and I am sure that the book will be very helpful not only to the undergraduate students in dermatology but to the general medical practitioners in the tropics

Department of Medicine
R G Kar Medical College,
Calcutta
15th August, 1956

PREFACE

With the slow recognition of skin diseases as a speciality in the tropics the dearth of a book on tropical dermatology has been realised both by the medical students and the practitioners With a view to help them I have attempted to give a basic idea of the skin diseases in the tropics by writing this 'Treatise On Tropical Skin Diseases'

This book has been prepared on my twenty years' experience in dermatology both in the tropics and in Europe and with the help of my lecture notes, series of articles published by me in the Indian Medical Forum (Calcutta), my knowledge gained from the pioneer dermatologists in Europe, from current medical literatures as well as from the books by different authors on the subject of dermatology to all of whom I acknowledge my gratefulness For writing the foreward I am thankful to Dr Amal Kumar Roychoudhury, Director Professor of Medicine and Principal, R G Kar Medical College, Calcutta

Suggestions for improvement will be received thankfully and for errors, short comings and omissions I regret very much

To Messrs Tacker, Spink & Co, (1933) Private Ltd Calcutta I am grateful for taking all the responsibility to print and publish the first edition of my book.

43, Dharmatala Street,
Calcutta-13, (India)
30th September 1956

KALI DAS LAHIRI

CONTENTS

A Treatise on Tropical Skin Diseases

CHAPTER I.
INTRODUCTION

Man has admired the beauty of the skin since the beginning of the universe. The beauty is said to be skin deep but the skin diseases are deeper still. Poverty, worry and sorrow would age a person and no cosmetic can restore the beauty. Thus beauty depends on heredity, age and healthy body

Developmentally the dermis and the hypodermis develop from the **mesoderm** while the epidermis develops from the **ectoderm** of the embryo

The skin covers the whole body and thus is one of the vital organs of the body The skin is modified to the needs of the part of the body concerned. It is thin all over with rugosity over the scrotum and thickened over the palms and soles The skin is covered with thick hairs on the scalp, axilla, pubic region and on the perineum but the rest of the body is covered by



Fig No I
Section of Skin
(Skin of an adult Indian)

lanugo hairs In the male beard and moustache grow on the face. Hairs, nails and sweat-glands are the secondary appendages of the skin

The **histology** of the skin is important (Fig No 1) The true skin is called the dermis or cutis The portion of the skin above the dermis is called epidermis and the portion below the dermis is know as hypodermis

The epidermis of the skin has five different layers showing different characters of the cells (Fig No 2)

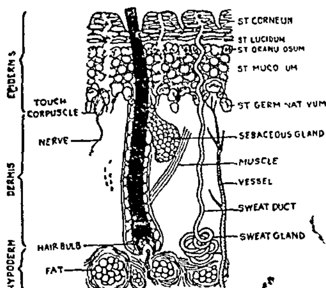


Fig No 2

Section of Skin (Diagramatic)

The layer which is just above the dermis is called the stratum **basalis** or stratum germinativum The basal layer consists of one layer of cylindrical cells Each cell has thin protoplasm and a nucleus Each nucleus has a melanin cap at the top which protects the nucleus from the damaging effect of the sun's rays Some of these cells become specialized and take up special function of forming the pigment of the skin which is called melanin

These specialized cells of the stratum basalis are called **melanoblasts** A number of fine dendritic processes arise from the body of the melanoblast which get connected to the neighbouring cells of

stratum basalis Through the dendritic processes melanin pass from the melanoblast to the cell which gets arranged like a cap on the top of the nucleus These melanoblasts look black when stained with Bloch s dopa called Dopa positive and also look black when stained with ammoniacal silver nitrate From the basal layer other layers of the epidermis are formed As the basal layer grows old another basal layer grows beneath it and the older layer is pushed up Several of these old basal layers go to from the stratum **mucosum** or prickle cell layer, which is also known as rete mucosum Usually five layers of cells go to form the stratum mucosum In this layer the cells are polygonal with prickles over their surfaces. Prickles are fine strands connecting one cell with the other Protoplasm is said to flow through these prickles form one cell to the other The protoplasm in the cell of the stratum mucosum is slightly thicker than that of the cells of the stratum basalis The nucleus is present but there is no melanin New stratum basalis is being formed continuously and the cells above it are pushed up When the cells of the stratum mucosum are pushed up the top layers become flattened Usually two layers above the stratum mucosum show changes The nucleus becomes shrunken The protoplasm becomes very much thickened and is called **keratohylin** Protoplasm looks granular and the nucleus cannot be seen amidst the granules This layer is called stratum **Granulosum** When the stratum granulosum is further pushed up the cells are further flattened Generally two layers of cells above the stratum granulosum thus get changed and is called stratum **lucidum** which looks like a white streak under the microscope No nucleus can

be seen and the protoplasm changes into an oily fluid called eleidin. The two layers of dead cells above the stratum lucidum go to from the stratum **corneum.** The protoplasm is further changed into a substance called **Keratin.** There is no nucleus in the cell of stratum corneum. Stratum corneum is dry and hard due to the presence of keratin. The protoplasm of the cells in the epidermis is transformed into keratohylin in the stratum granulosum, to eleidin in the stratum lucidum and finally to keratin in the stratum corneum.

Corium or dermis is the true skin The upper half of the corium lying apposed to the epidermis is called the **papillary** layer of the corium and that below it is the **reticular.** The junction of the papillary corium and the epidermis is called the **dermo-epidermal junction,** which is represented by a wavy outline. The papillary layer is projected upwards by finger-like processes The portions of the dermis which lie at the top of these finger-like processes is called the **papillary bodies.**

A **papillary body** consists of a part of dermis, blood-vessels, lymphatics and nerve terminals.

The whole of the dermis consists of collagen fibres and elastic tissues. Blood vessels entering the dermis from the hypodermis divide and get arranged into two plexuses. The upper plexus is called the **papillary** plexus and the lower one is called the **reticular** plexus. There are veins, lymphatics and nerves travelling along with the arteries. The nerve terminates at the dermo-epidermal junction in the touch corpuscles called the **Paccinian corpuscle** and the **Meisener corpuscle.**

The hypoderm lies below the dermis. It contains fat cells enclosed in the connective tissue bundles. The hair-follicles and the sweat-glands lie in the h

SKIN APPENDAGES

The hair and the nail are the appendages of the skin.

Hair-Follicle—The hair lies enclosed at one end in a cylindrical structure and together with it forms hair-follicle or pilo-sebaceous follicle (Fig. No. 3). There is only one hair in a hair follicle The lower end of the hair follicle is dilated and is called the hair-bulb The hair blub lies in the hypoderm The body of the hair-follicle passes through the dermis and ends in the upper layer of the epidermis. The hair-follicle is composed of stratum corneum and stratum mucosum Hairs are being constantly shed and are replaced by new hairs during life Regrowth of the hair requirs different time for *different* parts of the body. Average time for the scalp is 129 days, 92 days for the chin and 64 days for the eyebrows. There is some factor present in the hair-follicle which brings about the cyclic change in the hair. About 40 hairs are normally shed every day from the scalp. At birth there is lanugo type of hairs all over the body. Soon

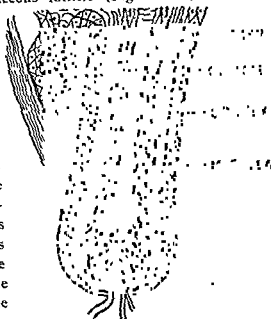


Fig No. 3.
(Diagramatic pilo-sebaceous follicle)

after birth hairs grow on scalp. At puberty hairs grow on moustache and beard regions in males and in the pubic region and axillae in both sexes. After the age of 40 the hairs grow on ears in males and in females at menopause there is generally growth of hair on the face The rate of growth of hair in a healthy body is about one fourth inch every *month.* The rate of growth of scalp hair is about ½ inch per month and the lanugo hair on other parts of body is about ½ inch per month. The growth of hair is not influenced by shaving. The rate of growth of the hair depends on (i) age, (ii) health, (iii) balanced diet, (iv) the use of *chemicals* for bleeching, dyeing, shampooing

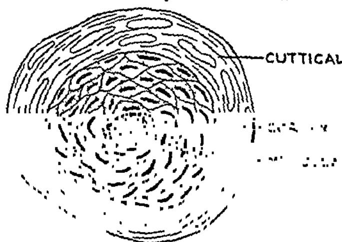


Fig. No. 4

Scalp hair of a Tropical girl (Hairs of an Indian girl from Bengal—5 ft. 2 inches in length)

and fixing hairs and also on (v) inherited factor. The hair may be straight or curling. Curling hair is seen amongst Negroes The colour of the hair differs in different races. Black is common in tropical countries Brownish and reddish colouration of the hair is not infrequently seen in many people in the tropics with fair complexion but is generally found in European races In India and other Eastern countries women and mendicants grow hairs on scalp The length of hair in Indian women is on an average 36 inches but sometimes hairs have been seen touching the heels (Fig. No. 4) In the tropics hair adds to the beauty of a woman.

The hair on section has three parts (Fig. No 5) The innermost part is the medulla, the middle portion is the cortex and the outer part is the cuticle.



SECTION OF HAIR
Fig. No. 5.

after birth hairs grow on scalp. At puberty hairs grow on moustache and beard regions in males and in the pubic region and axillae in both sexes. After the age of 40 the hairs grow on ears in males and in females at menopause there is generally growth of hair on the face. The rate of growth of hair in a healthy body is about one fourth inch every month. The rate of growth of scalp hair is about ½ inch per month and the lanugo hair on other parts of body is about ¼ inch per month. The growth of hair is not influenced by shaving. The rate of growth of the hair depends on (i) age, (ii) health, (iii) balanced diet, (iv) the use of chemicals for *bleeching*, dyeing, shampooing



Fig. No. 4

Scalp hair of a Tropical girl (Hairs of an Indian girl from Bengal—5 ft. 2 inches in length)

tion of its cells These glands are on the areola, the nipple, the axillæ, the genitals, the anus and develop at puberty Apocrine sweating can be caused by emotional stimulation as it is controlled by the autonomic nervous system

Axillary odour is due to the action fo the axillary micro organisms on the apocrine sweat. Inhibition of the growth of these organisms stops any odour formation in the axillæ. Children do not produce apocrine sweat and hence they have no axillary odour. Eccrine sweat forms no odour.

Nail—The nail is a solid plate It is a piece of highly cornified epidermis The three sides of the nail are inserted into the skin while the anterior end is free (Fig No 6) The whole of the nail is called the body. The whitish crescentic part of the nail which lies posteriorly is called the **lanula.** The two sides lie in a groove called the **nail groove.** The thin portion of the skin which covers the nail in the nail groove is called the **nail fold.** Below the lanula is the **nail-bed** consisting of several layers of polygonal cells and upon this the nail plate rests. The nail is produced from the nail bed. The body of the nail is formed of stratum

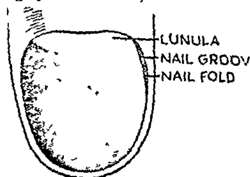


Fig No 6

A thin band of unstriped muscle fibres called **erector pile muscle** is attached to the hair follicle which by contraction erects the hair

Sebaceous Gland—Sebaceous glands are found near the hair-follicles The gland lies outside the hair follicle in an angle formed by the muscle of the skin with the hair-follicle. Two to four glands are found in connection with one hair follicle Each pours its secretion into the hair follicle The secretion of the sebaceous gland is called the sebum The sebum is a mixture of waxes and fats It lubricates, protects and anoints the skin surface

Sebaceous glands are found with the hairs of the scalp, axilla and the public regions There is another type of sebaceous gland which does not open into the hair follicle but opens directly upon the skin surface These glands are found about the lips, areola of the nipple and on the male and female genitalia

Sweat Gland—Is also known as the coil gland Sweat glands are found on the skin all over the body These glands have no connection with the hair follicles and open directly on the surface of the skin The sweat-gland is found in the hypoderm and the coiled tube passes through the dermis and epidermis and opens on the surface of the skin They produce colourless watery secretion to keep the body cool The sweat may be coloured due to parasitic infection and due to the ingestion of certain drugs

Sweat glands are of two types (1) Eccrine gland which produces fluid secretion These are found all over the body and its function is to cool down the body and the other type is called (2) the apocrine gland which produces secretion by the destruc-

sensible perspiration or sweating, (b) insensible perspiration is the loss of water from the skin surface and (c) mental sweating is the sudden appearance of sweating due to psychological stimulation such as are seen in sweating of palms and soles, (5) **Respiration** minute quantity of oxygen is absorbed through the skin in health, (6) **Sensation** the four important cutaneous senses are the appreciation of touch, pain, heat and cold Itching is also a type of pain sensation, (7) **Heat regulation** is done by the skin with the help of radiation, conduction and evaporation through the skin, (8) **Formation of VitaminD_2** by the action of the ultra violet rays of the sun on the steriod compound of the skin calciferol (VitaminD_2) is formed, which influences the calcium and phosphorous metabolism in the body, (9) **Metabolic function of the skin** is keratinization, (10) **Psychic function** emotional influence on the skin has been known long since. Fear is represented by blanching of the skin whereas shame by blushing, (11) **Disinfection** microbic infection is checked by the disinfecting power of the skin due to the acidity of the skin surface (which is pH 5 5) and the constant exfoliation of the epidermis.

pH of the skin—throughout life the pH of normal skin is 5·5 where eccrine glands are present but where apocrine glands are found the skin is pH 7 5 There is a definite relationship between pH values and the development of secondary sexual characters pH of axilla is 5.5 before puberty but becomes neutral after puberty

Body odour—the wild animals have a keen smelling power. The male follows a female only by the body odour which is left in the foot print on the dust. The odouriferous glands in the fourth interdigital space

lucidum. The rate of growth of nail is 0.119 mm. per day and there is no seasonal variation. Nail grows less in old age.

Skin Colour—The colour of the skin and hair is due to: (1) a colouring pigment called **melanin**, (2) vascularity of the part, (3) the thickness of the epidermis, (4) the relative luminosity, (5) melanoid, (6) hæmoglobin and (7) carotin.

Melanin pigment is formed by a complex bio-chemical process in some of the specialized cells of the stratum germinativum called **melanoblast cells.** These melanoblasts develop very late in the intra-uterine life and the complete development takes place after birth according to the physiological requirements The production of the melanin pigment depends on : (1) physical influence such as sun's rays, ultra-violet, heat, X-ray radiation, friction, (2) endocrine activity such as gonads, adrenals and pituitary gland, (3) vitamin dificiency such as A C and B-Complex, (4) nervous system such as sympathetic predominance inhibits the formation of melanin while the parasympathetic predominance *stimulates the formation of melanin.*

Functions of the Skin—(1) **Protection**: The skin protects the underlying soft tissues from acids, alkalies by the development of keratin in the stratum corneum of the epidermis and also by the formation of melanin in the stratum basalis it gives a protection against the heat of the sun, (2) **Excretion**: sweat-glands and sebaceous glands excrete various substances from the body such as sodium chloride, fats, fatty acids, cholesterol, and secretes moisture to keep the skin moist and smooth while the sebaceous glands scerete oil to keep the skin greasy, (4) **Perspiration**: is of three different types such as (a)

CHAPTER II

PATHOLOGY OF THE SKIN

By virtue of its position skin is exposed more to trauma and the finer variations in the skin pathology can easily be observed On proper recognition of this pathological changes the diagnosis and the treatment of the patients with skin diseases depend 'The essential changes are however, similar and in the skin are to be found congenital malformations hyperaemia anæmia inflammation hypertrophy and atrophy, where new growths abound and parasites flourish

The skin shows both (1) Primary lesion and (2) Secondary lesion

I **Primary skin lesions** are those which appear at the early stage of the disease

(a) **Macule**—Is an alteration in the colour of the skin of any size and shape as a result of pigmentation, depigmentation or dilatation of cutaneous vessels Examples are birth mark measles macular syphilide

(b) **Papule**—Is a small elevated lesion of the skin of any shape The epidermis gets thickened with vaso dilatation and perivascular infiltration in the dermis Papule may be shotty or India rubbery to the feel Seen in syphilis, lichen planus

(c) **Nodule**—Is a dermic swelling As in nodular syphilis leprosy

(d) **Tumour**—Is a larger dermic swelling e g melanoma

(f) **Plaque**—Is a localized atrophy or hypertrophy in the skin with or without scaling Examples are scleroderma lichen simplex

of each foot in the human being have atrophied. The body odour at the pubertal age is due to the activity of the glands in the skin. The sebaceous and apocrine glands become mature and produce secretion only after puberty in the human being. This body odour becomes aggravated during menstruation in the female and in seborrhoic subjects in both sexes.

various conditions In the stratum corneum the bulla is formed in impetigo and in scabies But in scabies there are two bullæ one in the stratum corneum and the other in the stratum mucosum like a dumb bell In the stratum mucosum again when a bulla is found at the upper part it may be due to herpes simplex or eczema when in the middle of stratum mucosum it may be due to herpes zoster or varicella while in the lower part of the stratum mucosum the bulla may be due to epidermolysis bullosum or pemphigus Sub epidermal bulla is found in dermatitis herpetiformis where the bulla develops under the stratum basalis and above the dermis

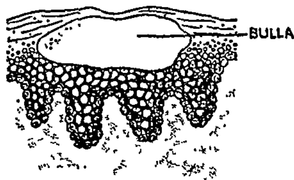
Cyto diagnosis Is done by preparing a smear on a glass slide from the floor scraping of an unruptured bulla and staining with leishman stain or Giemsa's stain The cytology of the bulla floor is helpful in the diagnosis of various skin diseases Large number of eosinophil cells with clumps of normal epidermal cells are found in the floor of dermatitis herpetiformis Enlarged giant epithelial cells called baloon cell are found in the scraping examination from the bulla floor of herpes simplex herpes zoster and varicella Absence of cellular elements is seen in the cytological smear examination of epidermolysis bullosum Lysis of the epidermal cells with pyknotic nuclei are found in the cytological smear examination of a pemphigus bulla (Tzanck test) In erythema multiforme floor smear examination shows large number of polymorphonuclear leucocytes with normal cells in the epidermis Tzanck test is helpful to differntiate pemphigus by the presence of its acantholytic cells from dermatitis herpetiformis and epithelioma

(g) **Wheal**—Is a transitory elevated red les with a whitish centre Seen in urticaria

(h) **Vesicle**—Is a circumscribed pin-head si swelling in the skin containing fluid S in eczema and pompholyx.

(i) **Bulla** - Is a circumscribed swelling in epidermis containing fluid and is larger in s than a vesicle Examples are herpes simp herpes zoster, impetigo, pemphigus.

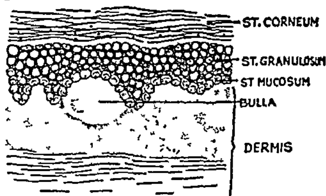
The situation of the bulla in the skin is help in the microscopical diagnosis of a skin disease Br may be (a) intra-epidermal (Fig. No 7) and (b) subepid



BULLA HIGHER UP IN EPIDERMIS

Fig No 7

mal (Fig. No 8) Intra-epidermal bulla is found



SUB-EPIDERMAL BULLA
Fig. No. 8.

The Secondary skin lesions are formed as a result of the degeneration of the primary skin lesions due to bacterial invasion and other causes —

(a) **Scales**—Are the dry exfoliation of the epidermal layer of the skin It is seen in ringworm, pityriasis versicolor and psoriasis Due to intra epidermal edema there is interference with the cell nutrition of the epidermis The stratum granulosum is incompletly developed and the cells are incompletly keratinized and the nuclei persist in the cells The process is called *parakeratosis* Is seen in eczema and psoriasis When there is localized or diffused hypertrophy of the stratum corneum it is called *hyperkeratosis*

(b) **Scab**—Is a dried mass of serum with scales, blood cells and organisms It is seen in impetigo

(c) **Excoriation**—Is the condion of the skin caused by the removal of epidermis from the skin Is seen in abrasions

(d) **Fissure**—Is a linear excoriation deep down to the dermis and is painful Seen at the angles of the mouth and in the groins.

(e) **Ulcer**—Is a circumscribed lesion characterised by the loss of epidermis and a portion of dermis It is seen in varicose ulcer.

(f) **Scar**—Is the new formation of fibrous tissue in the dermis There is no scar formation when the epidermis only is involved Scar tissue has no hair follicle

(g) **Pigmentation**—Occurs in the skin as a result of inflammation due either to increase in the melanin or due to extravasated blood.

(j) **Pustule**—Is a bulla filled with pus. A pustule is formed generally in association with a hair. The pustule may develop at the opening of a pilo-sebaceous follicle as in Bockhart's impetigo and at the bottom of a hair-follicle as in furunculosis. When many such hair-follicles are affected there occurs a phlegmonous degeneration forming multiple pus points like a honey-comb. Such a condition is called a carbuncle

(k) **Abscess**—Is a large pustule. There are two different types of abscesses: such as (i) ordinary abscess which is but a large sized pustule and the other is (ii) the micro-abscess which can be seen in the stratum corneum in psoriasis and is called micro-abscess of Munro, and also the intra-epidermal abscess of pemphigus vegetans.

(l) **Cyst**—Is a non-inflammatory pustule. When filled with sebaceous material it is called a sebaceous cyst.

(m) **Comedo**—Is a black grain-shaped plug at the mouth of a hair-follicle which is made of sebum and cells of stratum corneum. Comedo is seen on acne vulgaris.

(n) **Burrow**—Is a tunnel formed by the female sarcoptes scabiei in the layers of epidermis above the rete mucosum. The dead acarus is always found at the bottom of this tunnel. This is typical of a scabies lesion.

Collagen fibres or the elastic fibres may degenerate

(ii) **Allergy**—Is the specific hypersensitiveness of an individual to foods drugs bacteria and physical agents Allergy is a biological reaction of the body due to some skin disease and this allergy is an alterd capacity of the tissues to react to a substance Skin is an indicator of systemic allergic diseases

Various skin diseases are due to allergy Specific hypersensitiveness is always hereditary **Atopic dermatitis** is an example of such a specific hypersensitiveness in an individual The sensitizing substances are termed **atopens**

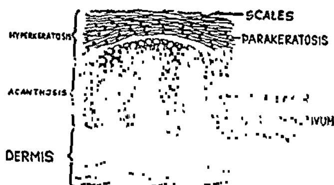
Body may be sensitized to any substance with which it comes in contact like chemicals proteins plants or flowers and sometimes to infection such as pyogenic infections and to fungus or tubercular infections When the body is sensitized by contact or by absorption there develops on the skin a rash which is called the 'id reaction or secondary rash

In case of bacteria it is called **bacterid,** in case of fungus it is **trichophytid** in case of tuberculosis it is called **tuberculid** This id reaction is a specific allergic sensitization to some toxin which is absorbed in the circulation

Types of allergy (1) **Food allergy**—some individuals are sensitive to certain food stuffs This specific hypersensitiveness to food stuffs is inherited in that particular individual Types of foods that cause sensitization are protein foods non protein foods organic foods and inorganic foods Food sensitization is commonly seen during the second year of life This type of sensitization to foods is a factor in the production of atopic eczema

(h) **Hyperkeratosis**—Is the thickening of the stratum corneum e.g. corns. (Fig. No. 9.)

(i) **Parakeratosis**—Is the presence of incompletly cornified, edematous and nucleated cells in the stratum corneum e. g. Eczema, psoriasis (Fig. No. 9).



THICKENING OF SKIN LAYERS

Fig. No 9

Section of Skin (Diagramatic)

(j **Acanthosis**—Is the thickening of the stratum mucosum e.g. warts, psoriasis (Fig. No. 9).

(k) **Lichenification**—Is characterised by papular, smooth and variously shaped skin lesions aggregated together with exaggeration of the normal skin markings showing a criss-cross pattern of the lesion. Develops as a result of long-continued itching e. g. Lichen simplex chronicus (Widal).

(l) **Atrophy**—Is characterised by the thinning of the epidermis. May result from wasting diseases, due to old age, lupus erythematosus, morphea etc.

(m) **Dermic degeneration**-Is of four types e. g. fatty, hyaline, mucoid and edema' 'e

(8) Blood sugar estimation (fasting)

(9) Blood cholesterol estimation (fasting)

(10) Blood calcium estimation

(11) Blood Vitamin A or C estimation (fasting).

(12) Blood protein estimations

(13) Mantoux test

(14) Sensitivity test

(15) Skiagram of chest and bones

(16) Biopsy and histopathological examination Biopsy material is preserved in a 10 per cent formaline in normal saline solution

(17) Skin scraping is examined for fungus with a 10 p c aq sodium hydroxide sol on a slide with a cover slip which is warmed on a Bunsen burner and the nail clippings are boiled with 40 per cent sodium hydroxide in a test tube for fungus examination

(18) Culture of skin scraping and nail clippings for fungus or bacteria in different media

(19) Cytology—fluid or the scraping of the floor of an unruptured bulla is examined for different types of cells In Tzanck test the smear on slides is stained with Giemsa s stain when acantholytic cells are seen in case of pemphigus

(20) Serology—blood W R and Kahn tests

(21) Animal parasites such as the louse or its eggs and the sarcoptes scabei are removed and are put on a glass slide with a drop of saline and are examined under microscope

Hence crab, prawn, egg and such other foods are discarded in patients suffering from atopic eczema

(2) **Drug allergy**—Some individuals are sensitive to some drugs Drug allergy is commonly seen in arsenic, sulpha-drug, antibiotic and such others Hereditary factor for this specific hypersensitiveness is present The mechanism of drug allergy is anaphylactic in nature. Drug allergy is specific and is permanent. Quite a large number of skin diseases are due to drug allergy. Dermatitis venenata, contact dermatitis, dermatitis medicamentosa and fixed drug erruptions are examples

(3) **Bacterial allergy**—fungus infection, pyogenic bacterial infection, syphilitic infection and tuberculous infection in a person may develop from sensitization in an individual

(4) **Physical allergy**—When symptoms are caused by cold, heat and light they are said to be due to physical allergy

INVESTIGATION OF A CASE OF SKIN DISEASE

(1) *Examination of stool after a saline purgative.*
(2) Examination of urine
(3) Blood picture—Total R B C and W. B C, differential count and hæmoglobin p c
(4) Erythrocytic sedimentation rate
(5) Examination of sputum
(6) Skin snip smear *examination* for *Bacillus* Hansen, Leishman donovo
(7) Nasal smear for Bacillus Ha

CHAPTER III

CARD OF A CASE

Date of first visit | Disease
Name | Address
Age | Sex
Referred by Dr
Complaints with duration
History

Family History
Personal History—Past
Present

General Examination—Hairs on head rash on forehead eyelids face and neck Jaundice or anaemia Tongue teeth, cervical axillary epitrochlear and inguinal glands and nails

Respiratory system
Cardiovascular system

Skin lesion
Investigation

(1) Examination of Urine
(2) Examination of Stool
(3) Examination of Blood for total differential counts, Hb p c and parasites
(4) Erythrocytic sedimentation rate
(5) Skin scraping examination under microscope for fungus or A F B
(6) Skin scraping for culture
(7) Blood chemistry
(8) Blood proteins
(9) Blood vitamins
(10) Biopsy
(11) Mantoux test
(12 Skiagram of chest and bones

Treatment
Follow up

(22) Diascopy—when a glass slide is pressed on a skin lesion the part is exsanguinated. Apple-jelly appearance is seen in case of lupus vulgaris on diascopy.

(23) Wood's light—the fluoresence is caused on the nail, skin or hair when exposed to the ultra-violet light which is filtered through a glass plate containing nickel oxide. This Wood's light is of special value in the diagnosis of different types of ringworm infections. Infected hairs with Microsporon fluoresce green whereas hairs infected with Trichophyton do not fluoresce. Wood's light is also used to examine the urine of porphyrin which gives a pinkish to red fluorescence.

CHAPTER III

CARD OF A CASE

Date of first visit | Disease
Name | Address
Age | Sex
Referred by Dr
Complaints with duration
History

Family History
Personal History—Past
Present

General Examination—Hairs of head rash on forehead eyelids face and neck Jaundice or anaemia Tongue teeth cervical axillary epitrochlear and inguinal glands and nails

Respiratory system

Cardiovascular system

Skin lesion

Investigation

(1) Examination of Urine
(2) Examination of Stool
(3) Examination of Blood for total differential counts Hb p c and parasites
(4) Erythrocytic sedimentation rate
(5) Skin scraping examination under microscope for fungus or A F B
(6) Skin scraping for culture
(7) Blood chemistry
(8) Blood proteins
(9) Blood vitamins
(10) Biopsy
(11) Mantoux test
(12) Skiagram of chest and bones

Treatment

Follow up

CHAPTER IV

TREATMENT OF SKIN DISEASES

In the tropical country it is always essential to treat the patient for any parasite in the gastro intestinal system e g amæbiasis giardiasis, oxyuriasis, ascariasis bacillary dysentry ankylostomiasis etc, blood infections like malaria etc, other infections like visceral kala azar, tuberculosis, nutritional deficiencies and blood diseases

Internally :

(1) (a) Arsenic—Liquor Arsenicalis (Donovon's solution) dose m 5, may be used in increasing doses (b) Pentavalent arsenic for injection such as Acetylarson, N A B Thiosermine (Brahmachari)

(2) (a) Penicillin crystalline 'G' aquous solution is injected intramuscularly in dose of *200,000* I U (0 2 mega unit) twice daily for 5 to 7 days, (b) Penicillin orally is some times used but does not have much effect in acute conditions, (c) Penicillin locally should never be used as it produces sensitization

(3) Streptomycin gram 1 by intramuscular injection Where patient cannot tolerate it Dihydrostreptomycin gram 1 is injected together with PAS by mouth

(4) Aureomycin (250 mg) capsule is given orally 6 hourly for 4 days usually with Vitamin B Complex by mouth 3 to 4 times after food

(5) Terramycin (250 mg) is given orally every 6 hours for 4 days with vitamin B Complex

(6) There are other antibiotics also e g Ilotycin Acromycin etc

(7) BAL (British Anti Lewisite) known as Dimercaptol 2 5 mg per Kilogram of body weight is used by intravenous injection This may also be used as a 3 p c ointment

(8) Antimony as stibanate (Gluconate) intramusculary or Ureastibamin (Brahmachari) intravenously or Stibinol (Brahmachari) or Stibatin (Glaxo) are used by intramuscular injections

(9) Sulphonamide such as Sulphadiazine, Elkosin etc, 0 5 gram is given orally every 4 to 6 hours for 4 to 7 days May also be given intramuscularly in acute conditions

(10) Sulphone such as D D S (B C P W), D A D P S (I C I), Novotrone (Bengal Chemical) Sulphetrone (B W), Thiosemicarbazone (A D) 50 mg daily, Diasone (Abbott) 0 3 gm daily as tablets are given orally in graduated increasing doses Injections of sulphetrone or novotrone solution are given intramuscularly

(11) Vitamins may be used together or separately by oral method and also by intramuscular route in high dose

(12) Liver extract is used both orally and intra muscularly

(13) Iron as Ferrous Sulphate is given by mouth

(14) Emetine hydrochlor is injected in one grain dose intramuscularly for 6 consecutive days with rest

(15) Enterovioform (Ciba) Enteroquinol (East India Pharm Works), Siosteran (Geigy) Aralis (Winthrop) are given by mouth

(16) Methionine as Neomethidine (Neo Pharma) is used orally as tablet or syrup and as solution intramuscularly or intravenously by injection to improve liver *function*

(17) Cortisone and ACTH are used for fatal skin diseases, incapacitating skin diseases drug reactions erythema multiforme and rarely in psoriasis with arthropathies dermatomyositis and urticaria Should be used very cautiously This therapy should not be stopped suddenly as it produces withdrawal symptoms characterized by exacerbation of the original symptom Cortisone suppresses the internal *production of the adrenocortical hormone* causing adrenocortical deficiency

(18) Isoniazide (Isonicotinic acid hydrazide) is used in cutaneous tuberculosis It is relatively non toxic May be used in combination with other anti tuberculous drugs

(19) Tar is commonly used in treating skin diseases As an antipruritic in 2 per cent solution as Liq Picis carb detergens and may be used with Lotio calamine or with an ointment Crude coal tar as 10 p c crude coal tar in Acetone is used for painting lichenified areas Prag mater (S K F) Primo derma (Primco) may be locally used as antipruritic and also for treating lichenified lesions of the ...

(20) Refrigeration is done by solid carbon dioxide It is used from 1/2 to 2 minutes Commonly used for infective warts, moles, superficial rodent ulcers and the like

(21) Ultra violet light is used in the treatment of various skin diseases

(22) X ray therapy, both superficial and deep, is used in malignant and other skin diseases

(23) Radium, as needles or plaques, is used in malignant skin deseases and haemangiomas

(24) Electro cautery is used for the removal of warts, cutaneous tugs, and small malignant growths Electro cautry needle is used for electrolysis and is commonly used for epilation of hairs in hypertrichosis

(25) Hyaluraunidase is sometimes used in the treatment of keloid

(15) Enterovioform (Ciba) Enteroquinol (East India Pharm Works) Siosteran (Geigy) Aralis (Winthrop) are given by mouth

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(iii) Ichthyosis hystrix is shown by localized exaggerated hypertrophy with dryness of the skin The hypertrophic skin generally forms bands on the extremeties

Signs and symptoms Dry and rough skin is known as xeroderma Patients feel dry There is no sweating particularly in winter When this condition is exagge rated with the appearance of fish scale like (Fig No 10) condition of the skin of the whole body except the flexures sometimes with a streak of pigmentation at the fringe of the scales is called ichthyosis This condition gets worse during winter Some of the patients suffering from ichthyosis develop localized band of hyperkeratosis on the upper and lower extremities called ichthyosis hystrix Patients become usually dark



Fig No 10
Ichthyosis (Boy of 6 years)

Patients seek cold and dry corners in a room Some of these patients also suffer from night blindness Itching is sometimes a troublesome symptom Itching causes scratches on the skin through which pyogenic organisms gain entrance and infective eczema sets in

Diagnosis (1) History of dry skin since childhood, (2) Clinical examination reveals dry, pigmented skin with

Chapter V.

CONGENITAL DISEASES OF THE SKIN

1. Fctodermal defect
2. Ichthyosis
3. Epidermolysis Bullosum
4. Urticaria Pigmentosa
5. Ehler-Danlos Syndrome

ECTODERMAL DEFECT

These are as follows :—

(1) Pachyonychia congenita is the absence or defect of nails.

(2) Absence of sweat glands.

(3) Dental dysplasia.

(4) Presence of nevus.

(5) Cutis vertices gyrata.

(6) Supernumary finger and supernumary nipple.

ICHTHYOSIS

Definition : Ichthyosis is a congenital skin disease characterized by dry fish-scale appearance of the skin.

Etiology : Is quite. common in the tropics. Supposed to be hereditary as it is sometimes found in more than one member of a family. Recently it has been found to be associated with Vitamin A deficiency. *Acquired ichthyosis is found in patients suffering* form Hodgkin's disease and nutritional dificiencies. The disease commences at the prepubertal age. Both sexes are equally affected.

Varieties : (i) Xeroderma is a mild hypertrophic condition with dryness of the skin, (ii) Ichthyosis is characterised by hypertrophy of the skin with dryness, fish-scale appearauce and pigmentation of the skin,

Treatment Prophylaxis patient should stay in places of equable climate and must avoid severe winter and should take vitamin A rich food

General treatment consists of applying locally oil all over the body before bath as is the practice in the tropics Olive oil is helpful Some patients are happy with the local application of a mixture containing equal parts of glycerine and water

Vitamin A is given in high doses of 50 000 international units twice daily by mouth throughout the winter and thereafter once daily with short intervals

Diet should consist of carrots butter, ghee milk liver, meat etc

EPIDERMOLYSIS BULLOSUM

Definition Is a congenital skin disease characterized by the development of bullae over the joints of the extremities by the slighest injury

Etiology Is found in the tropics Found in several members in a family

This condition is supposed to be associated with faulty porphyrin metabolism Occurs in both the sexes Sometimes Vitamin C deficiency is associated also

Varieties (i) Simple type (ii) Dystrophic type

Signs and symptoms The simple type occurs after birth Bulla is found with very mild injury on any part of the body The condition gets well at puberty

fish-scale appearance, (Fig No. 11) (3) Defective dai
adaptation, (4) Low blood vitamin A, (5) Histopatholog



Fig No. 11
Ichthyosis (Boy of 12 years)

shows thinning of the epidermis with loss of wavy outline at the dermo epidermal junction. Atrophy of sweat glands. Thickening of the stratum corneum with absence or marked thinning of the stratum granulosum.

Differential diagnosis. (1) Tuberculoid leprosy, (2) Seborrhoic dermatitis, (3) Besnier's prurigo.

Prognosis. Is not good so far as the cure is concerned. Death is not the common termination of the disease. Vitamin A therapy improves the conditio

Diagnosis (1) History of the development of bullae even with very mild injury at the places of



Fig No 14
Epidermolysis Bullosum

trauma (2) Clinical examination reveals bullae formation pigmentation and hypertrichosis (3) Porphyrin estimation in urine shows increased porphyrin excretion (4) Histopathology shows intraepidermal or subepidermal bulla formation There is hyperpigmentation in the stratum basalis Dermis shows rupture of the elastic tissue

Differential diagnosis (1) Pemphigus (2) Drug rash (bullous type)

Prognosis Simple type gets well at puberty but the dystrophic type is never cured

Treatment Prophylaxis consists in careful handling of the patients to avoid injury or pressure over the traumatic areas and vitamin C (200 mg) 2 to 3 times daily by mouth can be given

Treat the ulcer when develops with 2 p c Hydrarg Ammon ointment locally

The **dystrophic type** shows bullae developing after injury or irritation on the legs (Fig. No. 12). and hands, toes, ankles, knee joints and fingers. Wrist and elbows are also involved. Repeated bulla formation with cezematization occurs at the finger ends (Fig. No. 13)



Fig. No. 12
Epidermolysis Bullosum



Fig. 13. Epidermolysis Bullosum

The bulla fluild looks serous or mixed with blood. Bullae get ruptured and leave scars, pigmentation and and cysts in groups. The bulla may appear inside the mouth also. Nail changes may be present. Nail becomes birttle, rough and discoloured. Hypertrichosis is sometimes an associated feature.

pigmented itchy macules all over the body (Fig No 17) (3) Histopathology shows infiltration with mast cells (mast cells are cuboidal in outline) in the dermis Excessive melanin pigment is found along the stratum basalis with thinning of the epidermis In the bullous type the bulla is situated intraepidermally and a dense mast cell infiltration is found in the dermis below the stratum basalis

Differential diagnosis (1) Urticaria (2) Fixed drug rash (3) Xanthoma (4) Incontinentia pigmenti



Fig No 16
Urticaria Pigmentosa
(Back view of a girl aged 7months)

Prognosis Patients suffer throughout childhood Sometimes subsides at puberty or in adult life but the pigmentation is permanent



Fig No 17
Urticaria Pigmentosa
(Boy aged 6 years)

Treatment Locally antipruritic lotions are used such as Liniment Calamine with 1 per cent Phenol or an ointment containing —

Locally hydrocortone gives temporary encouraging results and cortisone orally is helpful when the patient is in distress.

URTICARIA PIGMENTOSA

Definition : Urticaria pigmentosa is a congential skin disease characterized by chronic, itchy and pigmented papules on the skin.

Etiology : Is not unknown in the tropics. Cause is not known. Some believe that it is due to the disturbance of some endocrine glands. There are others who believe it to be a congenital blood dyscrasia. Both sexes are affected but is common in males. Age of onset is usually before the sixth month of life.

Signs and symptoms : Starts in the first year of life (Fig. No. 15). Starts as macular or papular urticaria which is itchy but later on redness subsides and yellowish or blackish pigmented macules persist. The lesions may occasionally be bullous or nodular.



Fig. No. 15.
Urticarla Pigmentosa
(Front view of a girl aged 7months)

Five types of lesions are seen :— (1) Macular, (2) Papular, (3) Maculo-nodular, (4) Nodular, (5) Bullous. At puberty it gets apparently cured but reappears after puberty. Distribution is all over the body (Fig. No 16).

Diagnosis : (1) History of urticarial wheals starting early in infancy, (2) Brownish or blackish

Diagnosis (1) History of hyperstretchability of skin and exaggerated movements of joints since birth, (2) Skiagram of long bony ends shows abnoramal epiphysial structure (3) Histopathathology shows excessive formation of elastic fibres in the dermis



Fig No 18
Cutis Hyperelastica
case of Major A N Chakraborty A N Banerjee and S Ghosh)

Differential Diagnosis Cushing s syndrome
Prognosis Is not a curable disease
Treatment No treatment is of any use

Acid salicylic	....	gr.10
Menthol	....	gr. 5
Vaseline alba	....	oz. 1

X'ray therapy is sometimes helpful.

Internally—Antihistamins may be tried. Autohaemotherapy starting with 5 c. c. blood intramuscularly twice weekly and increasing by 0. 5 c. c. to 10 c.c., 12 such are helpful. Vitamin C (500 mg.) intramuscularly is injected twice daily for a week or so also may help. Adrenocorticotropin (ACTH) therapy in 20 units aquous *solution by intramuscular injection* every 6 hours or ACTH gel 20 units twice daily gives temporary good result. DOCA (Desoxycorticostrone) 4 mg. as sublingual tablet daily for 6 months has been advocated.

EHLER-DANLOS SYNDROME

(OR CUTIS HYPERELASTICA)

Definition : Ehler-Danlos syndrome is a congenital skin disease characterized by hyperextensibility of the normal skin.

Etiology : Cause is not known. Sometimes more than one member in the family suffer. Both sexes are affected. Starts from birth.

Signs and symptoms : *The skin of any (Fig No. 18)* part of the body can be stretched to a great length. Joint movements are exaggerated (Fig. No. 19) and wrist and finger joints are hyperextensible. There may be found ecchymosis on the extremities. Hyperextensibility of the abdominal skin may be found. Fine perchment like appearence of the skin is seen on hands and feet. Pads develop on elbows and knees (Fig. No. 20). In women clitoris may be enlarged.

CHAPTER VI

NON-INFECTIOUS INFLAMMATORY SKIN DISEASES

Non infectious inflammatory skin diseases form a group with an allergic background and is characterized by inflammation and itching

Classification 1 Eczema
2 Dermatitis

ECZEMA

Definition Eczema is a non infectious inflammatory syndrome characterized by itching and inflammation of the epidermis

Etiology (1) Irritant may be a cause such as chemicals when it is called **contact dermatitis** or **industrial dermatitis**, (2) Allergen may be a cause. Allergen may be of vegetable origin or animal origin when it is called **allergic dermatitis**, (3) Internal toxin may cause such as from food or auto infection when it is called **internal toxic dermatitis**, (4) Physical causes may lead to eczema such as burns from any source and is called **dermatoses due to physical causes.**

Classification of eczema (Modified from Percival's classification)

1 Eczema due to burns
2. Eczema due to contact
3 Eczema due to infection

such as follicular, flexural and post traumatic

4 Eczema due to varicose vein
5 Atopic eczema

such as infantile eczema, Besnier's prurigo, nummular eczema and lichen simplex chronicus (Widal)



Fig No 19
Cutis Hyperelastica
(case of Major A N Chakraborty
A N Banerjee & S Ghosh)

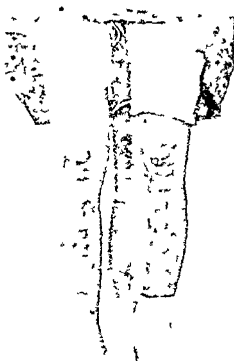


Fig No 20
Cutis Hyperelastica
(case of Major
A N Chakraborty
A N Banerjee and S Ghosh)

different chemicals in the laboratory of the medical chemistry, and in handling different medicines (Fig No 21)

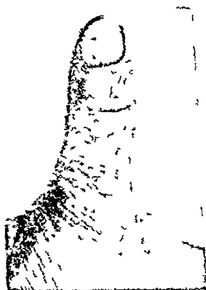


Fig No 21
Dermatitis
(Contact dermatitis due to hypo in a photographer)

in the practical pharmacy laboratory Common amongst the practising medical practitioners who have to handle different antibiotic solutions for injection Amongst the Indian ladies who put vermillion (Fig No 22) on their forehead as a cosmetic and those who use Sandel wood paste on forehead as a religious cosmetic (Fig No 23) or alta these types of chemical eczemas are of common occurrance (Fig No 24) Amongst ladies it is commonly found after the use of different cosmetics



Fig No 22
Contact Dermatitis Forehead
(Due to vermilion used as a cosmetic)

ECZEMA DUE TO BURN *It is not rare* Sunlight causes this type of eczema The fireman in the engine, the cook, those who handle ultra violet rays, X rays, radium, tracer substances are likely to get eczema due to burns Even when the skin is burnt by fire there may develop eczema The sunlight causes eczema alone when the skin is sensitized such as are

(a) *Solar eczema,*
(b) Hydroa aestivals,
(c) Xeroderma Pigmentosa,
(d) Drug sensitivity,
(e) Sun's ray is responsible for the production of certain diseases such as are
(i) Pellagra, (ii) Lupus erythematosus,
(iii) Sudamina, (iv) Keratosis

The lesions are erythematous which soon become *papulo vesicular in type Sometimes the lesions may* be bullous as in Hydroa aestivals

The skin lesion is very itchy and leave permanent scarring when incessantly scratched The most important predisposition to solar eczema is caused by the taking of sulpha drugs Pigmentary changes may also occur

Treatment Consists in avoidance of the direct exposure to sunlight and protection against ultra-violet, x-rays, radium and the like Workers who have to expose *themselves to fire should* be properly protected In a developed case Lotio Calamine when frequently applied helps *10 p c Para Amino Benzoic Acid locally may help*

ECZEMA DUE TO CONTACT It is very common all over the world and increases in a country with its industrialization *It* is seen amongst the non-clinical medical students who have *to handle* the formalised bodies while doing dissection, while handling

as lipstick rouge pomade snow nail varnish plastic as spectacle frame (Fig No 25) or as hand bags and



Fig No 25
Contact Dermatitis Face
(Due to use of plastic spectacle frame)

ornaments and is known as **cosmetic dermatitis** Use of chromium plated articles (Fig No 26) and handing



Fig No 26
Contact Dermatit s
(Watch band dermatitis due the use of chromium plated watch band)

chromium may cause contact dermatitis The workers in the industry have to handle different types of chemicals



Fig No 23
Contact Dermatitis Forehead
(Red sandal paste used as a cosmetic)



Fig No 24
Contact Dermatities Sole
(Due to cosmetic use of Alta)

(Fig No 29 & 30) which is becoming a problem in the developed countries of the world

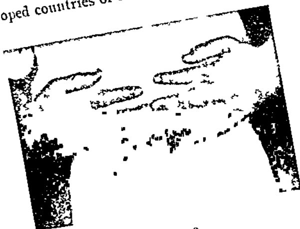


Fig No 9
Industrial Dermatitis
(Due to handling of va n l)

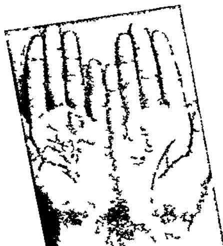


Fig No 3
Industrial Dermatitis Palms
(Due to handling of jute)

and oils which cause contact eczema and is called trade eczema or (Fig. No. 27 & 28) **Industrial Dermatitis**



Fig. No- 27
Industrial Dermatitis
(Due to plastic)



Fig. No. 28
Industrial Dermatitis
(Due to hypo in a photograph)

(Fig No 29 & 30) which is becoming a problem in the developed countries of the world



Fig No 29
Industrial Dermatitis
(Due to handling of varnish)



Fig No 30
Industrial Dermatitis Palms
(Due to handling of jute)

Any type of acid, any alkali and all the different salts either alone or in combination are liable to produce contact eczema. As an example carbolic soap may produce contact eczema. Wrist watch bands made of plastic material or chromium plating produce contact eczema.

Signs and symptoms. In the early stage edema, erythema and vesiculations with no margin can be seen. Generally seen on the uncovered parts of the body but may get generalised and can affect the scalp, eyelids and face. In the chronic stage it resembles chronic eczema characterized by lichenification with itching or erythemato vesiculation. Sometimes the parts become hyperpigmented and not infrequently leucoderma has resulted from this.

Diagnosis. (1) Careful history of work, hobby and the like, (2) Patch test is done using the suspected material. In a positive case an eczema reaction is obtained in *24 to 72* hours, (3) Biopsy and *histo-pathology* shows the formation of vesicle in the stratum mucosum with intra cellular edema of the epidermis and dilatation of the papillary blood vessels. Later on there is acanthosis with parakeratosis and hyperkeratosis.

Differential Diagnosis. In the acute stage contact eczema has to be differentiated from Erysipelas, Cellulitis, Erythema multiforme and Dermatitis herpetiformis.

Complication. Is the id reaction characterised by the development of an erythematous rash all over the body which on the palms and soles become vesicular. The distribution is symmetrical and is itchy.

Prognosis Is good when diagnosed and is treated properly

Treatment As a prophylaxis the offending material must be removed In the acute stage frequent application of (a) Lotio Calamine is very helpful Sometimes different types of lotions give good result such as

(b) Menthol	dr ½
Zinc oxide	dr ½
Glycerine pure	oz 1
Liq Plumbi subacetatis dil	oz 1
Liq Calcis	oz 20

Lotio for external use To apply every hour for a day or two

In the sub acute stage when there is much oozing soaks with 1 p c lotio silver nitrate frequently for 24 to 72 hours is helpful but when the oozing is non dripping 1 p c aquous gentian violet is painted When dry it is kept bandaged with 1 p c ung Ichthyol for about a week changing the dressings every day and cleaning with bland soap (Margo soap of Calcutta Chemical) and boiled warm water When chronic 2 p c Acid salicylic in a bland ointment (c) or 2 p c liq picis carb detergens or both may be used Bland ointment consists of

(c) Zinc oxide	dr 1
Pulv amylum	dr 1
Vaseline album	oz 1

Ung for external use

When dry and is left with a lichenified area 10 p c crude coal tar in acetone is painted once daily and is dusted over with a powder (Pearl powder—B C P W) and is kept bandaged for 24 hours with the bland ointment It is cleaned next morning with

warm water and a bland soap. After drying the part with spirit rectificatus coal tar is again painted and dressings applied. This treatment is *not* repeated for more than 6 times as tar is carcinogenic. Even after this treatment a remnant is left which is *treated* with suberythema doses of x-rays at fortnight's intervals with a low k v. Worker must be kept off work.

NAPKIN RASH

Definition : It is a skin disease of the infants characterized by erythematous or erythemato-squamous rash over the crural, genital, perineal and gluteal regions.

Etiology : It is a type of contact dermatitis. Alkali is supposed to be the cause The urea in the stool and urine of the infant ferments into ammonia and is said to cause the napkin rash. Sometimes it is thought to have a constitutional basis. Common *in infants for whom* napkin is commonly used. In the tropics napkin dermatitis is much less common as the napkin is changed soon after it gets soiled.

Signs and symptoms : *It starts* as an *erythema* but soon the lesion becomes papular and vesicular. After sometimes the vesicles rupture giving rise to multiple ulcers Site-it is confined only to the napkin area e.g, *in* the crural, genital, perineal, gluteal and sometimes the medial sides of both thighs The erythemato-papulo-vesicular rash may spread down the legs to the heels or upwards over *the back or abdomen.*

Diagnosis : (1) Erythemato-papulo-squamous rash, (2) Site-napkin area, (3) Age of patient-infant.

Differential diagnosis : (1) Contact dermatitis to soap, *(2) Congenital syphilitic cutis, (3)* Fungal infection.

Prognosis- Is good.

Treatment

As a prophylaxis a powder may be dusted over the napkin area containing Zinc oxide dr 4
Pulv Bismuth subgallate dr 2
Pulv. Calomel gr 15
Ft Pulv

Prophylaxis is to examine the napkins frequently and change it as soon as it is soiled After washing the napkin with soap and water it should be washed under running tap water and finally rinsed in a 10 p c aquous lotion of Boric acid and dried. Sometimes lotio for external use is helpful Acid Tannic gr 15 in aqua destilata oz 1 Ft lotio for external use or "Tanofax" ointment (B W) may be used

Curative consists of washing the buttocks and napkin areas with boiled warm water and drying the parts before putting on napkin again Dilute solution of tyrothricin has been advocated for local use on the skin lesions Orally a powder may be given twice daily for 4 days such as Grey Powder gr ½ Ft Pulv for a dose

ECZEMA DUE TO INFECTION

This type of eczema is caused either by strepto, staphylo yeast or fungus These are further classified under 3 groups such as—

(a) Flexural infective eczema,
(b) Follicular infective eczema,
(c) Post traumatic infective eczema,

Flexural infective eczema may be erythemato vesicular or vesiculo squamous lesions distributed on the flexural surfaces (Fig No 31) such as behind the ears, on the eye lids, below the chin in the axillæ, below the

pendulous breasts, in the umbilicus, in the groins, in the netal cleft and in between the fingers and toes



Fig No 31
Flexural Infective Eczema

In follicular infective eczema the hairy regions are involved such as the beard region when it is called "sycosis barbæ" (Fig No 32), the nuchal region when it is called 'sycosis nuche", pubic region and the limbs (Fig No 33) The Scalp of infants may be affected causing eczema of infants (Fig: No 34) which is different form infantile eczema.



Fig. No. 32
Sycosis Barbae

Rarely the follicular infetive eczema produces a



Fig No 33
Follicular Infective Eczema
(Dorsum of feet)



Fig No 34
Eczema of Infant
(Face is free)

band like lesion on a limb which may be linear or spiral in shape This type of eczema is known as **Lichen striatus**

Post-traumatic infective eczema is commonly seen amongst masons, miners, and amongst those whose works involve handling of dusts. Erythematous or erythemato-squamous lesions are found on the limbs extending from the ankles (Fig. No. 35) and



Fig No 35
Post-Traumatic Infective Eczema
(Showing chronic edema of one leg)

wrists to the knees and elbows. Post-traumatic infective eczema is also found on the nipples Eczema of nipples is common amongst mothers who are nursing their babies (Fig. No. 36).



Fig No 36
Eczema of Nipples and Breasts
(Case of Captain S N Roy)

Treatment Removal from the place of work is a prophylactic measure if it is a dusty occupation Treatment is given according to the stage of the disease If it is an acute condition lotio calamine is applied at frequent intervals In the sub acute stage when there is dripping oozing 1 p c aquous silver nitrate solution soaks are applied In the non dripping subacute stage 1 p c aqueous gentian violet is painted followed by 1 p c Ichthyol ointment and is kept bandaged In the chronic stage is used 2 p c ung Acid salicylic or 2 p c Liq picis carb det or both in a bland ointment containing Zinc oxide and Pulv Amylum each dr 1 in an ounce of vaseline alba This ointment is applied for a week Then if need be 10 p c crude coal tar is painted once daily for

4 to 5 days. At the end x-ray therapy for 3 to 4 times at fortnightly intervals will cure the condition

ECZEMA DUE TO VARICOSE VEIN

Etiology · The cause of the eczema is not known Inherent weakness of the venous system may be responsible for the eczema.

Predisposing causes in women are chronic constipation, pelvic tumour, pregnancy and the like Common amongst those who keep on standing long periods. Common in both sexes.

Signs and symptoms Tortuous prominent veins are seen on the calfs, on the knee joints and along the thighs. Oozing round about the ankles may be the earliest sign followed by erythemato-squamous lesions which on cleansing shows patchy denudation of the epidermis A punched out ulcer may be produced round about the ankle surronded with erythemato-squamous and eczematous lesion with pigmentation (Fig No. 37)



Fig. No. 37
Varicose Eczema

Prognosis is good

Treatment is by rest and elastoplast dressings to the varicose veins Injection of sclerosing fluid in the veins is also done

Eczema is treated with 1 p c aquous Silver nitrate solution soaks for 2 days and then by 1 p c aquous gentian violet followed by 1 p c ung Ichthyol and bandaged over with elastoplast dressing

ATOPIC ECZEMA . This is a constitutional skin disease The factors which are responsible for the causation of atopic eczema are heredity, over anxiety and a sense of insecurity. Onset or relapse of the disease occurs with the psychological upsetting Sometimes there is a hypersensitivity to some known allergens The allergy may be present to particular foods to different bacteria or to intestinal parasitic infections which are so common in the tropics

Predisposing factors are anaemia malnutrition and septic foci

Classification (1) Infantile eczema
(2) Besnier s prurigo
(3) Nummular eczema
(4) Lichen simplex chronicus (Widal)

(1) Infantile eczema It is an eczematous condition on the face of children occurring any time from the age of two months and disappearing by the age of two years

Signs and symtoms Starts as erythemato vasicular lesions on face (Fig No 38 & 39) but scalp may be



Fig No 38
Infantile Eczema



Fig No 39
Infantile Eczema

involved and later on it may spread over the trunk. Has a particular predilection for the face and flexural areas of the body Severe itching is present. The

vesicles get ruptured and erythemato squamous leasions develop with exudation and itching The patient is a well nourished child and the nutrition does not suffer There are periods of quiescence followed by exacerbation The exacerbation occurs particularly at the time of teething.

Diagnosis (1) Age of onset, (2) Well nourished child with eczema confined to face and flexural surfaces

Differential Diagnosis (1) Seborrhoic dermatitis, (2) Eczema in childhood, (3) Fungal infections

Prognosis Is good particularly with modern medicines But sudden death sometimes occurs Small pox vaccination should not be given

Treatment General—Antihistamins are given for 5 days by mouth such as Syrup Benadryl (P D), Antistin (Ciba), Pheuergan (M & B) Change of brand of antihistamins from Antistin to Avil or some other may produce better results. Adrenocorticotrophic hormone (ACTH) may be used by injection but has a temporary effect Cortisone orally may be used but the result is temporary Hydrocortone ointment may be used locally but the result is not permanent.

In the acute stage 1 p c aquous silver nitrate lotion as soaks are valuable but in the subacute stage Liniment calamine alone or with 2 p c Liq picis carb detergens may be used with benefit such as

Calamine ppt	dr 1
Olive oil	dr 4
Liq picis carb det	m 10
Aqua calcis	oz 1
Liniment for external use	

In the dry stage a tar ointment application with bandaging is necessary such as :

Crude coal tar	...	dr. 1
Zinc oxide	....	dr. 1
Pulv. amylum	...	dr. 1
Vaseline alba	....	oz. 1

Ung. for external use.

or Pragmatar or Primoderm may be used with benefit.

Diet is most important in infantile eczema. Milk and milk products should not be given. Sometimes fruit juice is also contraindicated.

Multivitamin and particularly vitamin C are given to the patient regularly Egg, lobster and crab are avoided in food

(2) **Besnier's prurigo** occurs after puberty usually in patients of infantile eczema and is characterised by erythematopapular lesions on the cubital and poplitial fossæ (Fig. No 40) and sometimes on the forehead and sides of the neck

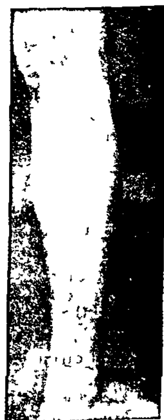


Fig No 40
Besnier's Prurigo

There may be acute exacerbation of the lesions followed by complete remission leavig only thickened skin in the cubital and popliteal spaces Itching is very severe and in acute stage there may be oozing

Treatment Change of environment is beneficial to help the psychological upsetting Patients are better treated in hospitals or nursing homes and should be kept away from relatives

Local treatment and general treatment are the same as for infantile eczema

X ray therapy is sometimes helpful

(3) **Nummular eczema** are erythematous, circular and coin shaped lesions on the extremities (Fig No 41) and are particularly on the extensor surfaces Itching is present and the skin is dry On the erythematous areas there is very slight sticky exudation present



Fig No 41
Nummular Eczema

Differential diagnosis (1) Psoriasis (2) Contact eczema, (3) Ringworm

Prognosis It is difficult to cure Relapses are common

Treatment: General treatment consists of giving orally Vitamin A in high dose (100,000 i.u.) for a long time. Locally when exudation is present 1 p.c. aquous gentian violet is painted followed by the application of 1 p c. Ichthyol ointment and keeping the part bandaged up. In the chronic stage tar is used as follows:

Zinc oxide	...	dr. 1
Pulv amylum	...	dr. 1
Liq. picis carb det.	....	m. 10
Vaseline alba	....	oz 1

Ung. for external use.

Sometimes crude coal tar is helpful. 10 p.c. crude coal tar in acetone is painted once daily and is dusted over with Pulv. Zinc oxide and Pulv Amylum in equal quantity and then kept bandaged with a piece of linen smeared with an ointment containing:

Zine oxide	...	dr. I
Pulv. amylum	...	dr. I
Vaseline alba	. .	oz. 1

Ung. for external use.

X-ray therapy sometimes gives good result.

(4) **Lichen simplex chronicus (Widal):** this is also known as neuro-dermatitis. Occurs in adults and particularly in those who shares heavy responsibility. The lesions occur usually on the nape of the neck (Fig. No. 42), side of the neck, round about the albow, medial side of the thighs and front of the ankle. Lesions are lozenge-shaped papules which coalesce together to *form* a plaque which feels thick and tough and is pigmented at the periphery with criss-cross markings. Lesions

are very itchy (Fig No 43). Due to scratching strepto and staphylo get entrance through the scratch marks and produce eczematous reactions at times It persists from 3 months to 30 years or more Rarely it spreads and covers the whole body and is called **lichen simplex chronicus disseminatus.**



Fig No 42
Lichen Simplex
Chronicus (Widal)

Prognosis Depends upon the psychology of the patient and proper handling of the case

Treatment Patient should be investigated psychologically and in resistant cases help of the psychiatrist is needed Phenobarbitone in grain 1 dose every night is helpful Largactil (M & B) 25 mg tab helps in dose of one tablet after food 3 to 4 times daily Locally when there is infection present it should be treated with 1 p c silver nitrate solution in water or 1 p c aquous gentian violet painting followed by the application of an ointment containing



Fig No. 43

Lichen Simplex Chronicus (Vidal)

1.	Zinc oxide	dr. 1
	Pulv amylum	dr 1
	Liq picis carb detergens ..	m. 10
	Vaselin alba	oz. 1
	Ung for external use twice daily	
or 2	Crude coal tar .	dr. 1
	Zinc oxide	dr. 1
	Vaseline alba	oz 1
	Ung for external use once daily	

or 3 10 p c crude coal tar in acetone painted once daily or Pragmatar or Primoderm 1% may be used locally

4 Hydrocortone ointment may be used but the result is not permanent

5 'F99 ointment may be used with temporary benefit

ECZEMATID

Eczematid reaction occurs as a secondary rash in association with a pre existing eczema The toxin is absorbed from the site of the lesion and is toxic in nature Lesions are pinkish and pin head sized when appears at first but soon becomes oval in shape with a diameter of ½ to 1 inch and become itchy The lesions are vesicular and bullous at times and are particularly so when appear on the palm and sole Eczematid may be localized or generalised When generalised it is symmetrical in distribution

Id reaction when occurs in association with eczema and is due to bacteria it is known as 'bacterid', when due to fungus it is called 'trichophytid', when in association with tuberculosis it is called 'tuberculid', when in association with syphilis it is called 'syphilid' Pompholyx is supposed to be a special type of 'id' reaction Some think that the origin of pompholyx is in the sweat duct

Differential diagnosis (1) Pityriasis rosea, (2) Seborrhoic dermatitis (3) Tinea corporis

Prognosis Is good

Treatment General treatment is to give an (1) alkaline mixture 4 times daily containing



Fig. No. 43

Lichen Simplex Chronicus (Vidal)

1	Zinc oxide	dr. 1
	Pulv. amylum	dr. 1
	Liq. picis carb detergens	m. 10
	Vaselin alba	oz. 1
	Ung. for external use twice daily	
or 2	Crude coal tar	dr. 1
	Zinc oxide	dr. 1
	Vaseline alba	oz. 1
	Ung. for external use once daily	

DERMATITIS

Definition The skin disease which is characterized by the inflammation of the dermis and is associated with itching and erythemato papulo vesicular lesion wi exudation and later crusting is called dermatitis

Classification (1) Drug dermatitis
(2) Artificial dermatitis
(3) Industrial dermatitis
(4) Seborrhoic dermatitis

DRUG DERMATITIS

Drug dermatitis is caused by the administration of one or more drugs

Etiology Hypersensitiveness to drugs is commonly seen in general practice and recently it has much increased due to the introduction of sulpha drugs, various antibiotics and continuous introduction of new drugs to the medical practitioners Sometimes a particular drug does not produce rash but when different drugs are used cross sensitization occurs and rash develops Sometimes inherited predisposition may be present

The predisposing factors to drugs are (1) increased susceptibility of the skin and (2) concentration of the drug in the skin

The increased susceptibility of the skin is due to the following factors such as

(a) Allergy which is a specific hypersensitiveness due to previous use of the drug The immunological mechanisms are responsible for this drug allergy

(1)	Pot cit	dr. ½
	Liq. ammon cit	dr. 2
	Syp. Aurantii	dr. 1
	Aqua menth pip.	ad oz. 1
	Ft. mist. for a dose.	

or Alkacitrone (Gluconate) 2 teaspoonfuls in water 4 to 6 times daily.

(2) Antihistamin is given for 5 days in the form of Antistin (Ciba) as injection one ampule intramuscularly every night and one capsule of Benadryl in the morning and another capsule at noon for 5 days either alone or together with injection.

(3) Vitamin C may be given orally in dose of 500 mg. thrice daily or by injection once or twice daily.

Locally may be used *Lotio* Calamine with phenol such as :

Calamine ppt. ..	dr. 1	
Phenol	dr. ½	
Aqua destil	oz. 8	Lotio for external use

To be applied every hour on linen for 2 to 4 days and treat the eczema also.

Lacto Calamine (Crookes), Caladryl (P.D.) or Calgesic (S. & D) may be repeatedly used locally.



Fig. No. 44
Drug Rash
(Due to paludrine by mouth)



Fig. No. 45
Dermatitis Medicamentosa
(Due to injection of quinine)

One type of allergic response is seen in some drugs while several different types are seen with other drugs.

(b) Idiosyncrasy is a peculiarity of an individual to develop drug rash even with therapeutic dose of a particular drug.

(c) Photosensitization is the provocation of a drug rash with actinic rays of the sun.

(d) Existing skin disease also helps to cause the development of drug rash.

(e) *Nervous irritability due to the toxic effect of the* drug on the nervous system.

The concentration of the drug in the skin as a result of (a) Preparatory to normal excretion—excretion is done through the sweat glands and sebaceous glands by exfoliation.

(f) Impaired excretion by kidneys and bowels. The mechanism of the production of the drug rash is not well-known. Many dermatologists consider the drug reaction to be an anaphylactic reaction.

The drug hypersensitivity is generally a permanent affair and the drug allergy is specific to a certain degree.

Drug rash *is classified as follows* :—

(a) **Dermatitis medicamentosa** is caused by the administration of a drug either by mouth (Fig. No. 44) or by *injection (Fig. No. 45). Examples* are *post-*arsenical dermatitis (Fig. No. 46 & 47), gold dermatitis, bromide and iodide rash and the like.

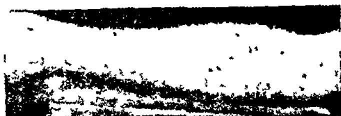


Fig No 44
Drug Rash
(Due to paludrine by mouth)



Fig No 45
Dermatitis Medicamentosa
(Due to injection of quinine)

One type of allergic response is seen in some drugs while several different types are seen with other drugs

(b) Idiosyncrasy is a peculiarity of an individual to develop drug rash even with therapeutic dose of a particular drug

(c) Photosensitization is the provocation of a drug rash with actinic rays of the sun

(d) Existing skin disease also helps to cause the development of drug rash

(e) Nervous *irritability due to the toxic effect of the* drug on the nervous system

The concentration of the drug in the skin as a result of (a) Preparatory to normal excretion—excretion is done through the sweat glands and sebaceous glands by exfoliation

(f) Impaired excretion by kidneys and bowels The mechanism of the production of the drug rash is not well known Many dermatologists consider the drug reaction to be an anaphylactic reaction

The drug hypersensitivity is generally a permanent affair and the drug allergy is specific to a certain degree

Drug rash is classified as follows —

(a **Dermatitis medicamentosa** is caused by the administration of a drug either by mouth (Fig No 44) or by injection (Fig No 45) Examples are post-arsenical dermatitis (Fig No 46 & 47) gold dermatitis bromide and iodide ras



Fig No 48
Fixed Drug Rash
(Vesicles on erythematous base)

found in a patient The lips may also show pigmentation The skin lesion caused by a particular drug gets exaggerated following the admistration of the same drug The earliest lesion may be an itchy erythematous plaque with a sharp margin There may develop vesicles on the erythematous area which later undergoes involution leaving a pigmented macular lesion The pigmentation remains for a long time. Drug rash is common in both the sexes and in all ages

Fig No 49
Fixed Drug Rash
(Due to Sulpha drug)





Fig. No. 46

Post-arsenical *Dermatitis*

(Due to injection of arsenic)

(Case of Major S C.P. Sinha)



Fig. No. 47

Post-arsenical Dermatitis

(Due to injection of arsenic)

(Case of Major S C.P. Sinha)

(b) **Dermatitis venenata** is caused by the local application of a drug on the skin. Examples are sulpha-drug rash, carbolic acid rash which develop after using *them as dressings.*

(c) **Fixed drug rash** is a *sharply* circumscribed eruption which recurs in previously reacting areas after exposure to a drug (Fig. No. 48). The commonest variety of fixed drug rash is an erythemato-squamous or hyperpigmented plaque. Size varies from ½ to few inches in diameter (Fig. No. 49). In shape it is generally circular or oval. One or several such plaques may be

Treatment Prophylactic is to remember that no dye, sulpha drug and antibiotic should be used locally

Before using a heavy metal or arsenic kidney function must be tested or the urine should be examined for the presence of albumin and casts The liver function test should be done when possible When facilities are available blood vitamin C estimation is done because vitamin C is the normal physiological detoxicating agent in the body Patients with low blood vitamin C usually develop drug rash

Curative treatment consists of (1) stopping the drug, (2) One dose of S S Mag sulph is given by mouth, (3) 25 p c glucose solution 25 c c to 50 c c is injected intravenously with or without vitamin C (500 mg) for two weeks (4) Vitamin C (500 mg) is given twice daily by intramuscular injection or thrice daily by mouth in tablet form (500 mg) for a week or 10 days, (5) Sometimes BAL or Sodium thiosulphate (Ametox sodium—May and Baker or Contramine—British Drug House) may be used by injection for 5 to 7 days (6) When the drug rash is very severe in nature ACTH aquous solution is injected 6 hourly in 40 units dose for 3 days and then ACTH gel once daily for the rest of the week Cortisone (25 mg) tablet may be used orally every 6 hours for 3 days 15 mg for 3 days 10 mg for 3 days and 5 mg for 3 days and afterwards 5 mg twice daily for a week During the corticotropine or cortisone therapy the patient should be without salt and must have vitamin B complex 4 times daily by mouth

Locally in the early stage Lotio calamine is applied every hour Sometimes Liniment calamine is helpful

Signs and symptoms: Sometimes the rash is *localized* to a particular region of the body while generalised rash may also occur. The rash may be a *temporary* erythemato-macular or papular lesion. Erythema-multiforme type of rash and urticarial may also occur. Bullæ can also be seen and even granulomatous lesions may develop *in chronic poisoning*. Hyperkeratosis of palms and soles may occur. Gangrene may also occur. Epithelioma has resulted as a complication in arsenic therapy.

Sometimes the rash develops within four hours after *the administration of the drug such as in quinine*, sulpha-drug and penicillin rash whereas at other time the drug rash appears after a long time from a few days to a few weeks after the administration of a drug. As a rule the drug rash appears suddenly and disappears suddenly after the stoppage of the drug.

Diagnosis: (1) Sudden appearance *of the* rash, (2) Distribution of the rash being symmetrical, (3) Character of the rash—polymorphic, (4) History of the patient of drug taking, (5) Patch test is sometimes helpful, (6) Biopsy—histopathology—In a fixed drug rash the stratum basalis will show increased melanin formation in the *melanoblast* cells and the presence of melanin-laden chromatophores may be found throughout the dermis. The papillary blood vessels are found dilated with perivascular *infiltration* with lymphocytes *and* histiocytes. In the granulomatous bromide rash known as bromoderma pseudo-epitheliomatous hyperplasia of the epidermis is seen with multiple micro-abscesses.

Prognosis: Good with modern drug *like ACTH and* cortisone.

Signs and symptoms Found on the body which is within easy reach of the patient's hands Legs (Fig No. 50) and arms and forearms (Fig No 51) are common sites Lesions are mostly found on the anterior surface of the body and on the face (Fig No 52) Lesions have sharp margins



Fig No 50
Artificial Dermatitis
(Below the left Knee)



Fig No. 51
Dermatitis Artifacta



Fig. No 52
Dermatitis Artifacta.

with or without 1 p.c. phenol. An *ointment* may be used containing :

Calamine ppt		dr. 1
Menthol	...	gr. 10
Pulv Amylum	...	dr. 1
Vaselin Alba	...	oz. 1

Ung. for external use.

Later *on olive oil with or without* 1 p.c. *Hydrarg* Ammon or 1 p.c. phenol may be used When there is oozing 1 p.c. Silver nitrate soaks are applied every hour for 2 or 3 days. When exudation has almost subsided 1 p.c. aquous gentian violet lotion is painted followed by the application of 1 p.c ung. Ichthyol. In chronic stage it is treated as chronic eczema.

Sometimes in very severe non-oozing type of drug rash Hydrocortone ointment locally may be used with benefit.

ARTIFICIAL DERMATITIS

This is also known as Dermatitis Artefacta, Dermatitis Autophytica, Dermatitis Factitia, Self-inflicted Erruptions, Feigned Dermatitis or Hysterical Dermatitis.

Definition : Artificial dermatitis is characterized by erythema, ulceration and even gangrene caused by the neurotic patients on their own bodies.

Etiology : Beggers usually produce ulcers on their bodies, on the arms or legs to rouse sympathy and the malingerers do it to avoid doing any work Patients are mostly females Persons of any age may have the disease but is common amongst young women

(b) Liniment calamine
Calamine ppt dr 1
Oil Olive dr 4
Aqua calcis ad oz 1
Liniment for external use.

(c) Ointment
Calamine ppt dr 1
Acid salicylic gr 10
Vaseline alba oz. 1
Ung for external use

Permanent dressings when left for 3 to 6 days with an ointment helps to heal the lesions

(d) Ointment
Calamine ppt dr 1
Pulv amylum dr 1
Liq Picis carb det m 10
Vaseline alba oz 1
Ung for external use

Sometimes these patients are very clever and intelligent and it becomes difficult to treat them at their places of residence It has often been found helpful to treat these patients in hospital under efficient nurses

INDUSTRIAL DERMATITIS

Also known as occupational dermatitis or trade eczema

Definition Industrial dermatitis is caused by the application of different chemicals, application of heat and due to mechanical causes such as by dust or friction to the skin while at work in an industry producing eczematous reaction

Etiology One per cent of workers usually suffers from industrial dermatitis (Schwartz) In the tropics the

Diagnosis : (1) The stigmata of sensitiveness must be sought for such as nail-biting, facial expression and the mental make-up are important factors, (2) Sites of lesions, (3) Absence of conjunctival and faucial reflexes, (4) Hypo-sensitiveness to pain near the site of lesion, (5) Hyperaesthesia is a common symptom, (6) Patient often predicts the sites of fresh lesions on the skin.

Prognosis : Complete cure is the rule.

Treatment : Prophylaxis-psychosomatic skin diseases have started receiving attention and a skin specialist should try to look to a patient from that angle also.

Curative—the environmental relationship with the patient should be carefully studied and the patient's socio-economic and domestic situations must be taken into account and hospitalization of the patient is helpful. Calming functional *irritability is important which may* be done by psychotherapy and by good understanding between the doctor and the patient. The doctor need be very sympathetic. Sedatives are helpful and as a routine Elixir Bromo Valerian dr. 1 with Elixir Vitamin B-Complex dr. 1 may be given after food 3 times daily for about 2 months. Largactil (M. & B.) is helpful. Sometimes Valerian (B. C. P. W.) is injected intramuscularly biweekly. Phenobarbitone is very helpful as a sedative when used for a long time. Locally may be used :

(a) *Lotio calamine—Calamine ppt.* ... dr. 1
Zinc oxide ... dr. 1
Glycerine pure ... dr. ½
Aqua calcis ... ad oz. 1
Lotio for external use.

industry Sometimes the patch test is negative to one particular substance but is positive to a mixture of two or more substances used in the industry

Prognosis Is good with treatment and with proper preventive measures Sometimes a change in occupation is needed for cure

Treatment Prophylaxis (a) Proper selection of the personale in the industry, (b) washing of hands and feet immediately after handling any irritant or using overalls and gloves, (c) Facilities to change clothings, bathing and proper ventilation of the factory are essential In tropical countries putting on electric fans or air-conditioning of factories may help in checking of various industrial dermatitis, (d) Educating the industrial workers by lectures, cinematography etc to avoid the industrial dermatitis should be instituted, (e) Barrier creams should be used to protect skin

General – Rest with treatment The skin lesion is treated as any eczema

If it is in the acute eczematous stage repeated local applications of lotio calamine is the best treatment When the condition is in the sub acute stage with oozing 1 p c lotio silver nitrate soaks are applied every *hour for a* day or two and then the oozing almost disappears In this stage 1 p c lotio gentian violet application once daily followed by the application of 1 p c ung Ichthyol and keeping the part bandaged repeating this daily for a week In the chronic stage a bland ointment with 2 p c Acid salicylic or 2 p c liq picis carb det or with both is applied daily and kept bandaged repeating this for about a week or so

number of cases are increasing with the growth of industry in the country. The irritants enter the body through the hair-follicles and sweat ducts but the fat-soluble *chemicals get absorbed* through the skin. Irritation produced on the skin either by the contact of chemical agents, physical agents or by mechanical means produces dermatitis which is designated as the nidustrial dermatitis. Workers may suffer in any industry. Even *the typist who is handling the type-tape or carbon* paper suffers. The painter who handles different chemicals, the mason's skin is traumatized by sand and cement, *the miner's skin also gets traumatized by coal dust.* Predisposing factors are many such as (1) history of allergy such as eczema, urticaria, asthma, drug idiosyncrasy and (2) oily skin and seborrhoic diathesis

Age – may occur at any age.
Sex—may occur in both sexes.

Signs and symptoms : Erythematous lesion is the earliest sign and itching of hands and body may be the earliest symptom. There may be erythemato-papular, *papulo-vesicular or pompholyx-like* lesions, ulceration and gangrene may occur. Rash may cover the whole body. There may be intractable itching. Scratching may be followed by oozing and then crusting. *Cellulitis* may occur with edema of face, hands and feet. There may be albuminuria and microscopic haematuria. (For pictures see eczema due to contact on page 40).

Diagnosis : (1) Worker in an industry, (2) Skin lesions on exposed parts of the body usually but may be over the whole body, (3) The skin lesion spreading far beyond the area of contact with the material in the industry, (4) Skin test with the material used in the

Urticaria is an allergic reaction due to the introduction of a foreign protein in the body of a sensitized individual, (d) Psychological disturbance may cause urticaria, (e) Hodgkin's disease leukaemia and reticulo sarcomas may be preceeded by urticaria.

Classification
(a) Urticaria papulosa
(b) Urticaria bullosa
(c) Urticaria gyrata
(d) Urticaria haemorrhagica
(e) Urticaria factitia or dermographism
(f) Angioneurotic edema or Quincke's syndrome
(g) Familial urticaria
(h) Urticaria pigmentosum

Signs and Symptoms Sudden development of wheals with intense itching which is **Urticaria papulosa** (Fig No 53). The wheal may be bullous when it called **Urticaria bullosa** When the lesions are very large and of policyclic in shape it is known as **Urticaria gyrata.** Sometimes there are haemorrhagic plaques in the



Fig No 53
Urticaria Papulosa
(Case of Dr B N Banerji)

TOXIC DERMATOSES

Toxic dermatoses are due to unknown toxins.

Classification : (1) Urticaria
(2) Prurigo
(3) Purpura
(4) Erythema multiforme
(5) Erythema nodosum
(6) Dermatitis herpetiformis
(7) Pemphigus
(8) Epidemic dropsy

URTICARIA—Definition : Is an acute or chronic disorder of the skin characterized by the development of wheals on the body with intense itching.

Etiology : Urticaria is due to allergic reactions. In an acute case of urticaria a positive skin test can be found whereas in a chronic case the skin test (Prausentz—Kustner reaction) is usually negative. Urticaria may be due to (a) external causes such as due to heat, light and cold. It is said that the patients have an antibody in their serum which reacts with the physical agents on exposure of the skin and develop urticaria. The stings of insects are the common causes of urticaria, (b) Internal causes such as are often due to the ingestion of some drug like aspirin, sulphonamide, injection of antibiotics and the like, (c) Parasitic causes such as in the tropics are intestinal infections like round worm, amaebiasis, giardiasis and the like and in malaria infection. Percival thinks that to precipitate an attack of urticaria the state of the tissues are more important than the noxious substance which is protein in nature.

The lesions are commonly pink coloured papules which suddenly appear and disappear in about 3 hours time Gradually the lesions disappear at longer intervals and in chronic cases it becomes persistent This may be acute or chronic Acute urticaria may be accompanied



Fig No 55
Angioneurotic Edema
(Case of Dr K O Kandhari)

with intense itching redness, nausea and vomiting Chronic urticaria is a recurrent condition. Dermographism may not be found in patients with urticaria

Diagnosis (1) Sudden appearance of pinkish papular lesions over the skin with itching which disappears after about 3 hours in acute cases but in chronic cases it is persistent (2) History of indiscretion to diet or administration of drug or serum injection (3) Histopathology Urticaria is a triple response to the effect of histamin-like substance produced by the tissue

wheals or separate from it, this is a rare type and goes by the name of **Urticaria haemorrhagica.** In some individuals the skin is so irritable that slight scratching causes wheals to develop when it is called **Urticaria**



Fig. No. 54
Dermographism
(*Case of Dr K. C. Kandhari*)

factitia or **Dermographism** (Fig. No. 54). When large swellings appear on different parts of the body and involve the eyelid or lip it is called **Angioneurotic edema** (Fig. No. 55) **Familial Urticaria** is found in several members of the same family and is often due to psychological upset. **Urticaria pigmentosa** *is a chronic* skin disease which starts within the first year of life and is *characterized by urticarial* pigmented macules for which see page 34.

(Winthrop) after food 3 times daily for 10 days but there are other oral preparations available in the market which have effect both on intestinal and extra intestinal parasitic infections During this ten day therapy the pH of the gastro intestinal tract is changed by giving a special diet consisting of Rice, Dahi (Butter milk) and boiled vegetables followed by normal diet with high protein Similarly for giardiasis atebrin tablets are given for 5 days in dose of one tablet after food 3 times daily

The blood of the patient is examined for total count of W B C R B C, Differential count, Haemoglobin per cent and for parasites If malarial parasites are found the patient is treated accordingly

Saline purgative like S S Mag Sulph Oz 1 is usually given in the acute cases but in chronic cases Mist Alba Oz 1 thrice daily for 3 to 7 days has been found more efficacious

Autohaemotherapy is helpful starting with 2 5 c c of blood and increasing by 0 5 c c upto 5 0 c c are injected biweekly I M for 6 to 12 such

Antihistamine drug is given in acute stage by daily intramuscular injection for 5 days but in chronic cases 50 mg orally thrice daily for 5 days is found to be effective When after 5 days therapy there is no response it is helpful to change the brand of the antihistamines Antihistamine drugs do not neutralise all the histamines produced in the body They act by blocking the receptors of histamine Urticarial wheals can be reduced or suppressed by antihistamine drugs and the underlying cause of wheal formation is not affected All the antihistamine drugs have the same essential action

damage. Edema in the dermis in all the tissues with dilatation of papillary vessels. In chronic cases there is found cellular infiltration with leucocytes and mast cells.

Differential Dignosis :

(1) Prurigo nodularis	(2) Drug rash
(3) Secondary syphilis	(4) Hodgkin's disease
(5) Leukaemia cutis	(6) Mycosis fungoides

Prognosis : Is good in acute urticaria. In chronic urticaria patients suffer for a long time. Angioneurotic edema causes swelling of eyelids, lips and even trachea producing breathing difficulty and may even cause death.

Treatment : Prophylaxis—In the tropics amaebiasis and giardiasis should be avoided by observing scrupulous cleanliness of cooking vessels and utensils and stool should be examined at regular intervals and treatment is taken when needed for intestinal infection. In a malarial place mosquito curtains should be used and prophylactic anti-malarial therapy should be given. All foci of infection should be investigated and should be treated. Patient should be investigated from the psychological point of view and rehabilitation of the patient is necessary as a prophylactic measure.

Curative—In the tropics the patient should be thoroughly investigated for gastrointestinal parasitic infections like giardiasis, amaebiasis, blantidiasis and the like and should be treated accordingly. Chronic amaebiasis is very intractable and the routine treatment should consist of (1) Six daily intramuscular injection of emetine hydrochloride gr. 1 with rest together with (2) one tablet of Entero-vioform (Ciba) or Entero-quinol (East India Pharma Works), Siostearn (Geigy) or Aralis

Quinine bihydrochlor gr 5 thrice daily, for a week is of value in urticaria associated with malaria in the tropics Colonic lavage with Condy's lotion (1 in 1000) or with normal saline is helpful in chronic urticaria and angioneurotic edema

Moccasin venom in dose of 0 1 c c (1 300 000 solution) at 7 day intervals and increasing by 0 1 c c upto 1 0 c c has been found beneficial in chronic urticaria of unknown cause

Locally warm water bath twice daily gives relief Bath may be followed by application of a powder consisting of

Pulv Camphor	dr	½
Pulv Zinc oxide	dr	1
Pearl Powder (Bengal Chemical)	oz	1
Ft Pulv for external use		

Lotion may also be used such as

(1)	Calamine ppt	dr	½
	Liq Picis Carb det	m	10
	Aqua Distil	oz	1
	Ft Lotio for external use		
(2)	Calamine ppt	dr	½
	Phenol	m	5
	Aqua Distil	oz	1
	Ft lotio for external use		
(3)	Calamine ppt	dr.	½
	Zinc oxide	dr	½
	Phenol	m	5
	Oil olive	dr	½
	Aqua calcis	oz	1
	Ft liniment for external use		

on urticaria. Antistin tablet may be given as one tablet thrice daily or Phenergan, Benadryl, Anthisan, Dibistin may also be used orally. Calciluvin (Bœhringer) by injection may be given I. M. daily. Benadryl or Antistin may be used as injection as one ampule daily for 5 to 7 days.

Sometimes the antihistamine drug *may itself act as an* allergen and produces shock which is called "histaminoid accidents." This reaction may occur during or after the treatment.

Cortisone may be given in 10 mg. dose every 6 hours. *Cortisone causes rapid disappearance of the urticarial* wheals and itching. It should not be used in acute urticaria where antihistamines can safely be used. For chronic urticaria cortisone may carefully be used when it is difficult to control the intractable urticaria

Vitamin C (500 mg) by intramuscular injection every 12 hours or by mouth in tablet form every 6 hours for a week are helpful in chronic cases. Sometimes a combination of Calcium with Vitamin C is helpful.

Ephedrine sulphate in 25 mg dose may be given orally once or thrice daily in urticaria and may be repeated 3 to 4 times in angioneurotic edema

Adrenaline *hydrochlor (1 in 1000) by intramuscular* injection in dose of 0.5 to 1.0 c. c. is particularly helpful *in acute and severe attacks of the disease.*

Thyroid extract gr. ¼ tablet daily for a week is sometimes *helpful in chronic urticaria.*

Ekzebrol (Tosse) when injected intravenously in gradually increasing doses helps to control the itching in urticaria and has a sedati

(3) Antihistamine ointments are sometimes used for antipruritic effects But epidermal sensitization often occurs by the topical use of antihistamines in the form of ointment Some dermatologists have found the combination of antihistamine orally and locally to be more effective in giving relief to the intense itching of urticaria as the antihistamines act by their central analgesic and local anaesthetic effect Neuropsychiatric investigation is needed when nothing helps the patient

Psychological investigation to find out the precipitating factors and psychotherapy are helpful in some intractable cases of urticaria.

Diet Is important in urticaria The taking of egg or drinking of milk or some other such foods sometimes cause urticaria and it is helpful to avoid such things during disease Elimination diet is helpful 'During the treatment the patient should be put on bland and lacto vegetarian diet Sometimes milk is also stopped and the patient is put on rice or wheat products and vegetables for sometimes and then is gradually taken to normal diet by adding one article every third day

PRURIGO

Definition Is a chronic skin disease characterized by itching and nodule formation some of which are excoriated and are found generally on the limbs

Etiology Cause is not known This is associated in the tropics with malnutrition and psychological upset Sex found in both sexes Age in people of past middle age but there is often a history of its onset early in l·f–

(4)	Calamine ppt.	....	dr. ½
	Zinc oxide	....	dr. ½
	Menthol	....	gr. 2
	Glycerine pure	..	dr 1
	Aqua calcis	..	oz. 1
	Ft. lotio for external use.		

(5)	Thymol	...	gr. 2
	Gl*ycerine*	....	m. 20
	Aqua calcis	....	oz 1
	Ft. lotio for external use.		

(6)	Lotio Niagra	...	dr. 3
	Oil Olive	..	dr. 3
	Aqua calcis	...	ad. oz. 1
	Ft liniment for external use.		

(7) Caladryl (Parke Davis) Calmitol lotion (Siegfried), Calgesic ointment (Sharpe & Dhome) when applied locally has soothing effect in urticaria

Ointment may be used such as ·

(1)	Calamine ppt.	..	dr. ½
	Zinc oxide		dr. ½
	Menthol		gr. 5
	Vaseline Alba		ad. oz 1
	Ft Ung for external use.		

(2)	Zinc oxide		dr. ½
	Camphor Pulv	...	gr. 12
	Menthol	.	gr. 3
	Oil Eucaliptol		m. 2
	Vaseline alba	.	ad oz. 1
	Ft. Ung for external use.		

extensor aspects of the extermities (Fig No 57) but may also be found on the trunk over the suprascapular



Fig No 57
Prurigo nodularis

regions (Fig. No 58) Starts late in life but usually after puberty and persists throughout life Patients cannot have



Fig Fo 58
Prurigo nodularis

Types : (1) Prurigo simplex.
(2) Prurigo nodularis.

Signs and symptoms : **Prurigo simplex** is quite common and are found in all ages and in both sexes in the tropics. These are characterized by papular lesions on the limbs, particularly over the gluteal regions, arms and thighs. Lesions are skin coloured and are itchy. Itching causes excoriation and relief. Starts early in life before puberty and rarely disappears in adult age. Prurigo simplex rarely becomes prurigo nodularis in adult age **Prurigo nodularis** is not uncommon in the tropics Is often found in elderly males. Lesion is nodular and in size that of a pea (Fig. No. 56). It is of skin colour and is very very itchy. Generally distributed over the arms and thighs (Fig. No. 56),



Fig. No. 56
Prurigo nodularis

Curative Antihistamine may be tried in dose of 25 mg. tablet 3 to 4 times daily for a week and then with a maintenance dose of 5 mg thrice daily for weeks Phenobarbitone may sometimes be needed orally in dose of gr ¼ to ½ once or twice daily Ekzebrol (Tosse) intravenously is helpful in some cases

Locally (a) antipruritic lotions may be used containing

(1) Calamine ppt	dr	1
Phenol	gr	10
Aqua distil	oz.	1
Ft Lotio for external use		

(2) Calamine ppt	dr	½
Sulpher ppt	gr	10
Liq Picis Carb det	m	10
Aqua Distil	oz	1
Ft Lotio for external use.		

(3) Antihistamine lotions may be used but sensitization may develop sooner or later with it (4) Caladryl (P D) and Calmitol (Siegfried) give temporary relief from itching

(b) Ointments may be used in from of

(1) Acid Salicylic	gr	10
Liq Picis Carb det	m	10
Vaseline Alba	oz	1
Ft Ung for external use		

(2) Antihistamine ointments may be used but there is a risk of sensitizing the patient.

good sleep and are so such mentally disturbed by *constant itching that they* develop suicidal tendency also.

Diagnosis : (1) Nodular itchy and excoriated lesions distributed over the extremities and on the back, (2) Nervous type of patient, (3) In children bed-itching is a common symptom, (4) Histopathology-*hypertrophy of stratrum corneum stratum* granulosum and stratum mucosum is seen in prurigo nodularis (Fig. No. 59) while in prurigo simplex there is dystrophy of the dermis.



Fig. No 59
Histopathology of Prurigo nodularis.

Differential Diagnosis : (1) Urticaria, (2) Drug Rash, (3) Scabies, (4) *Pediculosis*, (5) *Lichen planus nodularis*, ( ) Secondery syphilis, (7) Von Recklinghausen's disease.

Prognosis : Persists throughout life

Treatment : *Prophylaxis consists in removal* of the patient to a better surrounding and away from psychological and familial influences

Curat ve Antihistamine may be tried in dose of 25 mg tablet 3 to 4 times daily for a week and then with a maintenance dose of 5 mg thrice daily for weeks Phenobarbitone may sometimes be needed orally in dose of gr ¼ to ½ once or twice daily Ekzebrol (Tosse) intravenously is helpful in some cases

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Aqua distil	oz	1
Ft Lotio for external use		

(2) Calamine ppt	dr	½
Sulpher ppt	gr	10
Liq Picis Carb det	m	10
Aqua Distil	oz	1
Ft Lotio for external use.		

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Vaseline Alba	oz	1
Ft Ung for external use		

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Prognosis : Persists throughout life

Treatment : Prophylaxis consists in removal of the patient to a better surrounding and away from psychological and familial influences

Prognosis Good

Treatment Prophylaxis consists of taking balanced deit and avoiding infectious diseases

Curative Rest in bed Vitamin C (500 mg) intra muscular injection every 6 hours for 3 days and then twice daily Sometimes repeated blood transfusion is required Vitamin C with Calcium is also helpful

Sometimes antibiotic therapy is indicated such as crystalline Penicillin 'G' 5 lacs (0 5 Mega unit) intra muscularly in water every day for 5 days

ERYTHEMA MULTIFORME

Definition Is characterized by the recurrent occur rence of erythemato papular lesions on the body

Etiology (1) Virus is said to be responsible for certain types of erythema multiforme such as Stevens Johnson syndrome, (2) Herpes simplex virus is said to be responsible, (3) Virus pneumonia may be respon sible, (4) Toxin is responsible for other types of the diseases such as erythema iris This toxin may be bacterial or due to a drug, (5) Endocrine diseases are somtimes responsible

Age—Commonly affects children Seen in both sexes

Classification (1) Idiopathic type
(2) Multiforme type
(3) Stevens-Johnson type

Signs and Symptoms In the idiopathic type the lesions are erythemato papular and the situated on the

Surgical treament is helpful. 1 p. c. local infiltration with *novocaine of the skin* arond a nodule is anaesthetised and the nodule is excised and stitched. One or two such nodules may be taken out weekly.

X-ray therapy is not of much use in these patients.

PURPURA

Definition: Purpura is charactarized by haemorrhage in the dermis with itching.

Etiology: Types—(1) Idiopathic.
(2) Symptomatic.

Age—at any age. Sex—may be found in both sexes.

Signs and Symptoms: Idiopathic purpura is characterized by fever, gastro-intestinal disturbance, swelling of joints and haemorrhage in the skin. In symptomatic purpura there is also haemorrhage in the skin in association with other diseases such as herpes zoster, pemphigus, infectious fevers, during the toxæmic stage and Vitamin C dificiency stage. Recurrences are common. Sometimes only the ankles may be affected with lichenoid purpuric pigmentation without any blood change except showing sub-clinical avitaminosis C in adult males which is called pigmented purpuric lichenoid dermatitis.

Diagnosss: (1) Haemorrhagic patches in the skin of the body and are particularly confined to the ankles, (2) No blanching of the lesion on diascopy examination, (3) Examination of blood for total count of W.B.C. and R.B.C., differential count, hæmoglobin per cent, parasite and platelet count, (4) Histopathology shows deposits of haemosiderin in the dermis.

conjunctiva (Fig No 61) There may be rhinitis, urethritis and vaginitis. Is common in both sexes and



Fig No 61
Erythema multiforme.
(Showing lesions on lips and eyes)
(Patient - Meena Kum

can be seen in all ages Skin rash may appear several days after the illness

(b) Bahcet s syndrome is characterized by inflammation and erythema of the mucous membrane of conjunctiva, mouth and genitalia with fever There may be intense photophobia and even Keratitis with circumorbital pain Oral lesions are erthymatous with a red halo There may be pain in the tonsils and in the tongue. Common amongst children and is found in both sexes

(c) Reiter's syndrome is characterized by conjunctivitis, urethritis and arthritis. Seen in adults. *Found* in both sexes but common amongst females. Skin lesions are rarely seen when present the skin lesions are urticarial, erythematous or nodular in nature The

extremites and even on palms and soles. Some of the lesions are circular and are called **Erythema iris**. Sometimes bullæ are found. In the multiforme type the lesions are large in size and are characterized by erythemato-papular lesions covering the trunk or the extensor surfaces of the limbs (Fig. No. 60). Distribution is symmetrical. There may be ulceration of mouth with or without any rash on the body. Onset is rapid.



Fig No. 60
Erythema multiforme
(Erythemato-papular lesion on thigh)

The Stevens-Johnson type is characterized by fever with inflammation of the mucous membranes of mouth and conjunctiva. There are three varieties of Stevens-Johnson syndrome such as :

(a) Stevens-Johnson syndrome is also called *Ectodermosis pluriorificialis erosiva* where there is erythema and ulceration of the mucous membrane of mouth and

presents a relapse, (7) ACTH injection is sometimes helpful at 6 hours, 8 hours, 12 hours intervals of 40 units of the aquous solution and then ACTH gel 40 units at 24 hours interval and subsequently reducing the dose to 30 units, 25 units, 20 units and 10 units (8) Antihistamines are sometimes helpful in a dose of 50 mg at 6 hours interval for 5 days, (9) Hydrocortison ointment (Roussel) locally allays itching

Diet Bland diet consisting of liquids like milk, tea, fruit juice for a week then fish and meat can be added with bread and butter If cortisone is used salt-free diet should be given or K salt (Calcutta Chemical) may be added to food

ERYTHEMA NODOSUM

Definition Is a skin disease characterized by the development of symmetrical painful nodules on the legs below the knees with slight fever

Etiology (1) Tuberculosis may be the cause and may be the sign of activation of a latent focus, (2) Rheumatic origin is also suspected, (3) Pyogenic infection—may be associated with strepto and staphylo coccal infection of tonsils and sinuses, (4) Meningo coccus—erythema nodosum may be symptomatic of meningococcal infection of the cerebro spinal system, (5) Gonococci sometimes erythema *nodosum* may develop in association with gonococcal septicæmia (6) Leprosy—may develop in a patient suffering from lepromatous leprosy, (7) Syphilis—erythema nodosum has often been found in syphilitics (8) Drugs—erythema nodosum has often developed during or after therapy with certain

eruptions are found on hands, feet and genitals. The disease runs a long course of 4 to 6 months.

Diagnosis: (1) Recurrent occurrence of erythmato-papular lesion which are symmetrical and are mostly on the extensor surfaces of the limbs, palms, soles with or without fever and conjunctivitis, (2) Histo-pathology shows edema of the whole dermis including the vessels and lymphatics with dilatation of blood vessels and lymphatics. Round cell infiltration which are mostly eosinophilic in the early stage but later on changing to lymphocytes. In the erythema multiform bullosum type the situation of the bulla is intra-epidermal.

Differential Diagnosis: (1) Uurticaria. (2) Dermatitis herpetiformis, (3) Drug rash, (4) Pemphigus.

Prognosis: Is good in most cases. The eruptions subside in 2 to 3 weeks.

Treatment: Prophylaxis – not know yet.

Curative—(1) Rest in bed (2) Saline purgative (3) Alkali mixture 4 times a day with or without Sodi-Salicylas – gr 10 per dose.

(4) Aureomycin capsule (250 mg) orally every 6 hours for 4 days together with Elixir Vitamin-B-Complex, 2 tea-spoonful twice daily are helpful.

(5) Vitamin C (500 mg) is injected intramuscularly twice daily for 7 days together with 10 p.c. calcium gluconate solution, (6) Cortisone (25 mg) by mouth every 6 hours with vitamine-B-complex for 4 days and then gradually decreasing the dose before finally stopping the drug shortens the duration of the attack but never

presents a relapse (7) ACTH injection is sometimes helpful at 6 hours, 8 hours, 12 hours intervals of 40 units of the aquous solution and then ACTH gel 40 units at 24 hours interval and subsequently reducing the dose to 30 units, 25 units, 20 units and 10 units, (8) Antihistamines are sometimes helpful in a dose of 50 mg at 6 hours interval for 5 days, (9) Hydrocortisone ointment (Roussel) locally allays itching

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drugs like sulpha-drugs, antibiotics, bromides, iodides and the like, (9) Sarcoidosis may be associated with it, (10) Ulcerative colitis may give rise to erythema *nodosum*.

Sex-may affect both sexes. Age-may affect any age.

Signs and symptoms : Malaise or slight fever *may be present for a day or two before the* development of painful, dusky-red nodule half pea sized to four times the diameter of a pea. The lesions develop on the calf muscles and on the front of the legs below the knee joints. Lesions are tender and occur on both the legs. They are painful. Nodules never suppurate nor ulcerate. In 2 to 4 weeks time lesions *gradually fade away*.

Diagnosis : (1) Symmetrical dusky red, tender and painful nodules on both legs below the knees, (2) No tendency to ulcerate, (3) Blood W.R. and Khan tests are negative except *in cases associated with syphilis*, (4) Tuberculin test (Mantoux test) is possitive, (5) E.S.R. is high, (6) Biopsy—histopathology shows edema in the dermis with dilatation of blood vessels and infiltration with round cells and epitheloid cells. Rarely giant cells are seen.

Differential diagnosis : (1) Drug rash, (2) Gumma, (3) Erythema multiforme.

Prognosis : *Is good. Seldom there is a relapse.*

Treatment. (1) Rest in bed is very important, (2) Skiagram of chest to find out a tuberculous focus

for treatment, (3) Legs are wrapped with cotton wool and kept at rest (4) A mixture is given containing

Sodii Salicylas	gr 10
Sodii Bicarb	gr 15
Liq Ammon Cit	dr 1
Syp Calcii hypo	dr 1
Aqua menth pip	ad oz 1

Mft Mist for a dose Send 8 such Is taken every 6 hours (5) A saline purgative is given at the beginning of the treatment (6) Sulpha drug such as sulphadiazine tablet every 6 hours for 4 days may be given, (7) Penicillin crystalline G in aquous solution of 5 lacs daily is helpful (8) Vitamin C (500 mg) may be injected at 12 hours interval or vitamin C (250 mg) tablet is given by mouth every 6 hours for a week

DERMATITIS HERPETIFORMIS

Definition Is a bullous dermatitis characterised by grouping itching pigmentation and recurrences

Etiology It is seen in the tropics and form about 1 0 per cent of all skin cases Sex—occurs in both sexes Age—found in all ages but is common amongst children in the tropics

Cause is not known but some toxin is supposed to be responsible for its causation Some think that a virus is resposible for the development of dermatitis herpetiformis Pregnancy is a predisposing factor and the dermatitis herpetiformis which develops during pregnancy is known as **herpes gestationis** or hydroa gravidarum **Impetigo herpetiformis** is another grave and rare form of dermatitis herpetiformis which affect males and the females

Signs and symptoms: Eruptions are erythematous or bullous in type. Bullæ develop for several days with burning and itching.

They are grouped and hence known as herpetiformis. Lesions are symmetrical.

There is no erythematous halo round the vesicle.

Sites are sacral region, buttocks, round the elbows, knees and scapular regions (Fig. No. 62 & 63. In male children sometimes is seen on the genitalia. Itching is felt long before the eruption appears on the skin and itching is relieved after the rupture of the bullæ. When the ulcer (Fig. No. 63) heals pigmentation is left. Rarely the

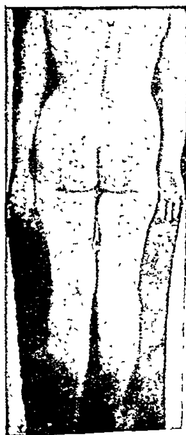


Fig. No. 62
Dermatitis herpetiformis



Fig. No. 63
Darmatitis herpetifor

mucous membrane is also involved Herpes gestationis develops early in pregnancy between the third and sixth months Lesions are erythematous papules or vesicles and are found in groups (Fig No 64) distributed on the



Fig No 64
Herpes gestationis

forearms, legs, buttocks, back and abdomen (Fig No 65). Eruptions disappear after the delivery. The child is



Fig. No. 65
Herpes gestationis

not affected by the eruptions *but stillbirths* and miscarriages may often occur. This is a recurrent condition.

Diagnosis (1) Symmetrical recurrent itchy grouped bullous lesions on sacral region and round the elbows and knees (2) Multiformity of lesions (3) Chronicity (4) Pigmentation (5) Eosinophilia in blood which may be above 30 p c and the variability of the eosinophil cells in blood on different days is peculiar in dermatitis herpetiformis (6) Cytology of a bulla will show over 30 p c of eosinophil cells (7) Biopsy—histopathology will show a subepidermal bulla and the presence of œdema in the dermis and epidermis Perivascular dense cell infiltration eosinophil in the dermis Hyper pigmentation below the stratum basalis is common

Differential diagnosis (1) Urticaria (2) Scabies, (3) Impetigo (4) Erythema multiforme (5) Congenital bullous syphilitic rash (6) Drug rash (7) Pemphigus

Prognosis Is good so far as the life is concerned in the younger age group There are always relapses of this condition and in particular during pregnancy In old age dermatitis herpet formis may change over to pemphigus when prognosis becomes grave Herpes gestationis does not affect pregnancy and gets well after termination of pregnancy

Treatment Prophylaxis investigation of all septic foci and their treatment are essential Patients develop ing herpes gestationis should avoid further pregnancies Rh factor of parents may be responsible for the develop ment of the disease in children

Curative—All hygienic measures should be adopted In the tropics cold water bath every day and warm water bath in winter is essential Bland soap may be used for bath

Arsenic is an old drug for this disease and is used in the form of liquor potassii arsenitis (Fowler's solution) given for a long time starting with one drop daily for a week, then increasing by one drop every week Acetarsone in dose of 0 25 gram 2 to 3 times daily for a week may be given Suramin (Germanin) or Bayer 205 may be used with success

For temporary relief 10 p c calcium gluconate may be injected intravenously

In herpes gestationis is advocated autohæmotherapy or injection of 10 c c of inactivated auto serum Progesterone injection has also been advocated in this condition Combined *cortisone and progesterone therapy* is also advocated, Proluton 50 mg daily for 7 days then 30 mg, 25 mg, 15 mg daily is sometimes valuable in herpes gestationis Cortisone (Roussell) 100 mg is given daily for 3 days, 75 mg. for 6 days and 50 mg cortisone is continued with 100 mg progesterone daily Autohæmotherapy with 10 c c of blood has also been advocated Subcutaneous injection of blood serum from healthy pregnant woman gives good results

Sulphapyridine (May & Baker) is given as ½ tablet every 6 hours for a week and then every 12 hours for a long time with regular checking of the blood picture Antihistamines may also be used for a week or 10 days Diazone or sulphoxone sodium (Abbott) in dose of one tablet every 8 hours for a week, later on twice daily for a week and is continued for a long time with one tablet every day keeping a check on the blood picture and *hæmoglobin* Dapsone or diamino phenyl sulphone is the drug of choice It is used in dose of 100 mg daily by

Antibiotics—Penicillin crystalline 'G' may be given by injection when scratching causes secondary pyogenic infection and systemic reactions Aureomycin may be used orally in dose of one capsule every 6 hours for 4 days together with vitamin B complex

Locally warm Condy's bath is helpful In severe itching Liniment Calamine with 1 per cent phenol is used Ointment may be used such as

(1)	Sulphur ppt	gr 20
	Zinc oxide	dr ½
	Vaseline Alba	oz 1
	Ung for external use	
(2)	Hydrarg Ammon	gr 20
	Zinc oxide	dr ½
	Vaseline Alba	oz 1
	Ung for external use	

(3) 3 p c Aureomycin ointment as well as other antibiotic ointments have sometimes been used

(4) Hydrocortisone (Roussell) ointment locally allays itching and produces soothing effect

Diet—Lacto vegetarian diet has proved of value in tropics Those who are non vegetarian it is advisable for them to avoid egg lobster and crab in food

PEMPHIGUS

Definition Is a chronic and relapsing skin disease associated with bulla formation on the normal skin and ending fatally sooner or later

Etiology: *Classification of pemphigus:—*

(1) Pemphigus acutus
(2) Pemphigus vulgaris inocuous
(3) Pemphigus vulgaris chronicus
(4) Pemphigus vegetans
(5) Pemphigus erythematode
(6) Pemphigus *foliaceus*
(7) Pemphigus neonatorum
(8) Ocular pemphigus
(9) Familial benign chronic pemphigus

Profession may be responsible for the acute type of the disease as it has been observed that those who *handle carcaces and butchers suffer from it.* Sometimes toxæmia is held responsible for pemphigus acutus. Pemphigus neonatorum is due to staphylococal infection of the skin in infants and is now called *Impetigo neonatoram.* Familial benign chronic pemphigus *is supposed to be a form of Darier's disease and* is associated with vitamin A deficiency. Cause is not known for other types of pemphigus. Some believe that the disease is a metabolic disorder. Virus is also held responsible for the causation of pemphigus.

Age—Acute pemphigus is common in young adults. *Pemphigus neonatorum is found in new-born* children. Other cases of pemphigus are *generally found* in elderly people past middle-life but in the *tropics it* affects round about the 40th year of age. Sex—affects both sexes but in the tropics seems to be common *amongst males.*

Signs and symptoms: ***Pemphigus acutus****—It is a* rare skin disease. Onset is sudden. *There is always*

present a history of having had a trauma during handling of dead bodies or having had vaccination Bullæ first appear on the neck and inside the mouth and spread rapidly all over the body (Fig No 66) in successive



Fig No 66
Pemphigus acutus

crops Bulla may be round like half hen's egg in size It may be *tense or flaccid* Sometimes bullæ coalesce together Contents of the bulla is serous to start with but becomes purulent later on Sometimes the bulla content may be hæmorrhagic also The bulla formation becomes generalised and covers the whole body including the mucous membranes of mouth, nostrils, eyes, rectum and in woman vagina Bulla ruptures soon leaving a raw and oozing surface

Temperature is very high even from the beginning, Patient generally becomes stuporose and dies within the first week of the attack Rarely the first attack subsides only to relapse after a month and the patient succumbs to it

Diagnosis (1) History of injury in a butcher or having had vaccination, (2) High temperature,

(3) Bulla formation, (4) Cytology of the bulla-floor shows no acantholysis, (5) Culture of the bulla fluid shows no organism by Gram's stain and most of the cells are polymorph with very few eosinophil cells if found at all, (6) Culture of bulla fluid is sterile, (7) Biopsy—histopathology of acute pemphigus is characterized by the formation of a bulla at the dermo-epidermal junction or in the middle of the stratum mucosum. Tzanck cells are also found which are characterized by the persence of rete cells in the vesicle.

Differential Diagnosis: (1) Dermatitis herpetiformis, (2) Erythema multiform bullosum, (3) Drug rash.

Prognosis: *Is very grave. Always ends fatally.*

Treatment: Prophylactic—to avoid injury in butchers and to dress surgically when there is any *trauma* in such a profession.

Curative—Aureomycin (250 mg.) capsule is given every 6 hours with vitamin-B Complex as a routine measure. Corticotropin (ACTH) aquous solution is administrad by I.M. injections in dose of 40 units every 4 hours for the first 2 days and then every 6 hours. *Blood transfusion is also given and may* be repeated daily if needed. Cortisone may be given (25 mg.) *tablet every 4 hours for 5 days and then* every 6 hours. Continuous warm water bath with dilute *Condy's lotion is very relieving.*

Diet—Liquid diet is given with high protein such as eggs, meat extractives, liver diet. Salt should not be given during ACTH or cortisone therapy. K-salt (Calcutta Chemical) may be used instead of ordi

(2) **Pemphigus vulgaris inocuous**—Is the benign type of pemphigus Patients are often admitted as cases of dermatitis herpetiformis but gradually develops the clinically developed stage of pemphigus vulgaris chronicus This type of pemphigus is commonly seen in the tropics Age—no age is exempt Sex—found in both sexes

Signs and symptoms few bullæ may develop on the normal skin (Fig No. 67) whithout any areola The



Fig No 67
Pemphigus vulgaris inocuous
(Case of Dr K C Kandhari)

lesions are half pea to half grape in size Lesions are often itchy Nikolosky s sign is present which is believed to be due to reduced calcium content of the epidermis It is a recurrent condition Leaves no scar or pigmentation on healing

Diagnosis (1) Recurrent type of bullous lesion which are itchy but leaves no scar or pigmentation on healing, (2) Nikolosky's sign is positive, (3) Biopsy—histopathology shows a subepidermal bulla (Fig No 68) at the beginning of the disease but later the bulla becomes intraepidermal (Fig No 69) with acantholysis, (4) Cytology shows acantholytic *cells* There may be round cells, most of which are poly but few may be eosino. The number of eosinophil cells do not increase on

subsequent examinations, (5) Bulla fluid-smear examination with Gram stain shows no bacteria, (6) Bulla fluid culture is sterile (7) Blood Wassermann and Kahn tests are negative, (8) Diminished output of urinary 17 Ketosteroid is considered to be an early diagnostic point

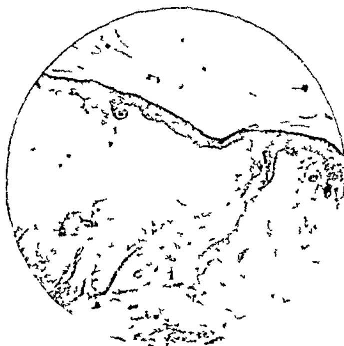


Fig No 68

Histopathology of Pemphigus vulgaris mocugus showing subepidermal bulla

(Case of Dr B N Banerji & Dr K D Lahiri)

Prognosis Fair but after several years develops into pemphigus vulgaris chronicus and the patient dies

Treatment Prophylaxis—nothing is known

Curative—Sulphapyridine tablet is given as one tablet 4 hourly for a week Aureomycin (250 mg) capsule is given with Vitamin B complex every 6

hours for a week. Autohæmotherapy—starting with 5 c.c. of patient's own blood on alternate days increasing by 0 5 c.c. every time until 10 c.c. is given.



Fig. No 69

Histopathology of Pemphigus vulgaris ii ocuous showing subepidermal bulla later becoming intraepidermal

(Case of Dr. B N Banerji & Dr. K D Lahiri)

Vitamin C (500 mg.) is given by injection twice daily and Vitamins A and B Complex are given by mouth Cortisone (25 mg) tablet is given by mouth every 6 hours for 2 days then reduced to 10 mg every

6 hours and ultimately 5 mg. 6 hourly, 8 hourly, 12 hourly and once daily before it is stopped. Corticotropin (ACTH) is given in the gel form in dose of 20 units twice daily for seven days and then gradually reduced to 10 units twice daily, 5 units twice daily and then once daily before it is finally stopped One or two blood transfusions are helpful.

(3) **Pemphigus vulgaris chonicus**—may occur as it is or may develop from a case of Pemphigus inocuous. Age—may occur at any age. Sex—found in both sexes. Quite common in the tropics.

No cause is known.

Signs and symptom : Sudden development of one or many bullæ on the normal skin. There is no areola round the bulla. *In size it may be half* pea to half grape or hen's egg. Bulla may be tense or flaccid (Fig. No. 70). Nikolosky's sign is positive. *Bulla fluid* is serous to start with but becomes purulent soon. Sometimes bulla fluid is also hæmorrhagic in nature. Situation of the bullæ are in the axaella, groin and may be anywhere on the body and mucous membrane. Sometimes the buila on the palate and tongue may appear first. Bullæ may be found in the nostrils and in the vagina and rectum causing prolapse of the uterus and prolapse of rectum also. When the bulla ruptures *a raw oozing surface* is left which is very tender and crust may form. Patient may develop fever late in the *disease and loose weight*

Diagnosis: (1) Sudden appearance of bulla with positive Nikolosky's sign,

peculiar mousey smell of the patient (3) Biopsy—histopathology shows an intraepidermal bulla with acantholysis



Fig No 70
Pemphigus vulgaris
(Case of Dr B N Banerji & Dr K D Lahiri)

(Fig No 71) (4) Cytology of the bulla floor shows acantholysis of cells There are round cells which are polymorphonuclear in type but few eosinophil cells may be found, (5) Bulla fluid shows the presence of no organism with Gram s stain (6) Bulla fluid in culture is sterile, (7) Serum protein estimation shows low albumin and high globulin Total protein is low (8) Blood Wassermann and Kahn tests are negative (9) Diminished urinary output of 17 Ketosteroid occurs quite early in disease

Differential Diagnosis (1) Dermatitis herpetiformis, (2) Drug rash, (3) Secondary syphilis



Fig No 71

Histopathology of Pemphigus vulgaris showing bulla in the epidermis and round cell infiltration in the dermis

(Case of Dr B N Banerji & Dr K D Lahiri)

Prognosis Is grave Patient dies within 2 years Rarely survives 8 to 10 years with treatment

Treatment Prophylactic—nothing is known.

Curative—Suramin is injected intravenously on alternate days in dose of 0 2, 0 3, 0 4 0 5, 0 6, 0 7, 0 8, 0 9, 1 0 g, 1 0 g

Acetarsol orally every morning for 3 days, rest for 3 days and repeating for 3 days, in dose of 0 25 g tablet

Aureomycin (250 mg) capsule is given every 6 hours with Vitamin B complex for a week or so or Chloramphenical 0 5 g 4 times daily with Vitamin B-complex for 2 weeks Cortisone tablet (25 mg) by mouth every 6 hours with Vitamin B complex orally for a week is helpful and then gradually the dose is reduced ACTH may be given in dose of 40 units of aquous solution every 6 hours for a week and then 20 units ACTH gel may be given twice daily for 4 days and then gradually reducing until stopped Bayer 205 by intravenous injection biweekly or Suramin may be given a trial Repeated blood transfusion (250 c c) weekly or biweekly is helpful

Pemphigus vegetans—Etiology Is very rare but they are seen in the tropics Both sexes suffer but is common in women

Signs and symptoms Malaise and often slight fever preceeds the formation of bullæ The onset is insiduous in pemphigus vegetans Bullæ are generally seen inside the mouth lips and nose first but may appear on the body before they come out inside the mouth The lesions on the body are seen around the genitalia, groin perineum exælla, extremities and on the scalp. The bulla ruptures and the base of the bulla becomes papular and gets covered by a crust Sometimes groups of blebs are seen which rupture and the bases proliferate to form vegetating masses (Fig No 72) when it is clinically a case of pemphigus vegetans Nikolosky's sign is present Gradually the patient looses weight Death is due to some intercurrent disease

Diagnosis (1) Formation of bullæ in the inguinal, perineal and genital regions, (2) Vegetating type of

lesions, (3) Biopsy—histopathology shows imperfect keratinisation of the stratum corneum, acanthosis and



Fig. No. 72
Pemphigus Vegetans
(Case of Dr. B. N. Banerji & Dr. K. D. Lahiri)

edema in the stratum mucosum. Bulla is intraepidermal but may also be subepidermal. Papillæ are hypertrophied and micro-abscesses are found in the papillary bodies with eosinophil cells. There is a compact mass of infiltration in the dermis with eosinophil cells which is very characteristic (Fig. No. 73), (4) Cytology shows presence of eosinophil cells with acantholysis Tzanck cells also are found, (5) Bulla fluid smear examination after staining with Gram's stain will not show the presence of any bacteria, (6) Bulla fluid culture is sterile, (7) Blood protein—total protein is diminished very much Albumin

is decreased and globulin is increased, (8) Blood chemistry—sugar, chloride and calcium are lowered, (9) Blood Wassermann and blood Kahn tests are negative, (10) Diminished urinary output of 17 Ketosteroid is an early diagnostic finding



Fig No 73

Histopathology of Pemphigus vegetans showing intraepidermal bulla with thickening of the prickle cell layer and micro abscesses

(Case of Dr B N Banerji & Dr K D Lahiri)

Differential diagnosis (1) Condylomata lata, (2) Condylomata accuminata (3) Bromide and iodide (fungating) rash (4) Dermatitis vegetans, (5) Other granulomas of skin

Pragonsis Is fair so far as the longivity is concerned compared with other types but the patient succumbs to some undercurrent disease May change to P vulgaris.

Treatment: Prophylactic—nothing is known.

Curative—Arsenic therapy is used. Carbarson 0.25 g. tablet thrice daily may be given. Sulpha-drug such as sulphapyridine or sulphadiazine *may ba given by mouth* as one tablet 6 hourly for a week Antibiotic such as crystalline penicillin 'G' *in aquous solution is injected in* dose of 5 lacs daily for a week or so. Aureomycin capsule (250 *mg) every 6 hours together with Vitamin* B-complex is advocated for a week or ten days and then repeated after an *interval of a week.*

Locally when there are bullous eruptions present Liniment Calamine with 1 p.c. Hydrarg Ammon may be applied every hour. When the bullæ rupture 1 p.c. Gentian violet is painted followed by 1 p.c. Ung Hydrarg Ammon dressing may be given. Antibiotic ointment may be helpful.

Injection of 2 c c. Crude liver extract is given daily. Repeated blood transfusions (250 c.c.) biweekly is very helpful.

(5) **Pemphigus erythematode** *this is also known* as Suear-Usher Syndrome.

May occur at any age but is commonly found in young adult males in the tropics.

Signs and symptoms: Erythemato-squamous patches may occur *at intervals on the scalp, face or trunk in the* intrascapular region and on sternum. These bullæ rupture and a raw, eczematous area resembling lupus erythematosus or seborrhoic eczema may be seen. Sometimes minute vesicles are seen at the periphery

of the these eczematous lesions (Fig No 74). May develop into pemphigus vulgaris



Fig No 74
Pemphigus erythematode
(Case of Dr B N Banerji & Dr K D Lahiri)

Diagnosis (1) Erythemato squamous or vesiculo-squamous lesions on the scalp face, intra scapular region and sternal region, (2) Follicular plugging in the lesion (3) Nikolsky's sign is positive, (4) Biopsy—histopathology shows acantholysis of cells in the stratum mucosum Dyskeratotic cells are found in the stratum granulosum There is no follicular hyperkeratosis Liquefactive digeneration of the dermoepidermal junction is characteristic (Fig No 75), (5) Diminished urinary output of 17 Ketostroid is an early finding

Prognosis : is best amongst its types and life may be as long as 10 or 12 years. Prognosis is grave

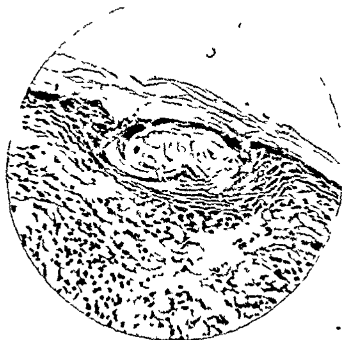


Fig. No. 75

Histopathology of Pemphigus erythematode showing bulla in the epidermis and round cell infiltration of the dermis and flattening of the papillae.

(Case of Dr. B. N Banerji & Dr. K. D. Lahiri)

when the lesions start *on the mucous membrane of* the mouth or change into P vulgaris.

Differential Diagnosis Lupus erythematosus, (2) Seborrhoic dermatitis, (3) Erythema multiforme.

Treatement . Prophylaxsis *is not known* Crude Liver extract injection and Vitamin A and C are helpful.

Curative—Antibiotics can be used orally and locally. Blood transfusion is indicated when condition becomes bad.

(6) **Pemphigus Foliaceous.** Etiology It is a rare disease Is also seen in the tropics. Affects adults Cause is not known

Signs and symptoms Bullæ appear which are flaccid in type Bulla spreads by peripheral extension Nikolosky's sign is positive Bullæ develop symmetrically and cover the whole body, extramities and face and even the mucous membranes of the eyes and mouth are involved Bullæ rupture and raw area is left Over the raw area flake like lesions develop The patient presents an exfoliative condition of the skin (Fig No 76). At intervals pemphigus foliaceous may change into pemphigus vulgaris chronicus



Fig No 76
Pemphigus Foliaceous
(Case of Dr B N Banerji & Dr K D Lahiri)

Diagnosis: (1) Exfoliative condition of the whole body, (2) Nikolosky's sign positive, (3) Biopsy—histopathology shows intraepidermal bulla formation which may be subcorneal. Acantholysis is commonly seen. Imperfect keratinization of the cells of the stratum corneum is seen showing parakeratosis. Acanthosis of stratum mucosum. There is edema in the dermis with round cell infiltration which are of lymphocytic, neutrophilic and histiocytic in types, (4) Blood picture shows eosinophilia, (5) Blood W. R. and kahn tests are negative, (6) Blood protein—albumin is decreased and globulin is increased, (7) Diminished urinary output of 17-Ketosteroid is an early sign of value.

Prognosis: is same as pemphigus vegetans.

Treatment: same as pemphigus vulgaris chronicus.

Locally Hydrarg Ammon ointment may be helpful. Blood transfusion is the sheet-anchor of treatment. Other treatments are supportive.

(7) **Pemphigus neonatorum**—Is impetigo contagiosum of the new-born (see impetigo).

(8) ***Ocular pemphigus***—*Is said to be a clinical* entity and is dealt in books of ophthalmology. In the *tropics almost every case of* pemphigus develops ocular lesions during the course of the disease (Fig. No. 77) but clears away with the improvement of the skin condition.

Prognosis: Does not become blind if properly treated.

Treatment: 2.5 p c. cortisone eye drop and eye-ointment are valuable Blood transfusion for the general condition helps to clear the eye lesions also.

(9) **Familial benign chronic pemphigus** is also known as Haily Haily disease—Is seen in several



Fig No 77
Pemphigus of the eyes
(Case of Dr B N Banerji & Dr K D Lahiri)

members of a family Deficiency of vitamin A is supposed to be the etiological factor Males are commonly affected

Signs and symptoms Recurrent bullous lesions develop on the sides of the neck and intertrigenous areas with positive Nikolosky's sign Mucous membrane lesions are not found Patient s health is not impaired

Diagnosis (1) Recurrent localised bullous rash on several members of a family, (2) Nikolosky's sign i positive, (3) Biopsy—histopathology shows dyskeratosi

of cells and partial acantholysis. Bulla occurs lower down in the stratum mucosum.

Prognosis: Is good.

Treatment: Vitamin-A in high doses when given for a *long time keeps the disease under control. Locally* 1 p.c. Hydrarg Ammon ointment is all that is necessary.

Pemphigoid—*The bullous rash* of *erythema multiforme bullosum* and *dermatitis herpetiformis* are sometimes called 'pemphigoid' from the benign nature of these rashes.

EPIDEMIC DROPSY

Definition: Is a tropical disease characterized by sarcoid-like lesions and with pigmentation and erythema over the skin accompanied with edema of legs and sometimes with fever and palpitation.

Etiology: This is due to cooking with mustard oil contaminated with argemone oil. The disease occurs in epidemic form at different places in the tropics. Age—people *of all ages are affected,* Sex—both sexes are affected.

Signs and symptoms: Starts as a gastro-intestinal disturbance followed by fever. Pallor appears together with engorged neck veins and edema of legs. Gradually multiple telangiectasia-like spider nævi appear all over the *body. The skin shows alternate* bands of erythema and blanching on pressure with spread-out fingers. Erythematous gyrate patches are seen (Fig. No. 78). Hyperpigmentation of the face and extremities are commonly seen. *Pin-head to pea-sized nodules and*

sarcoid-like lesions appear on the scalp, face and body



Fig. No. 78
Epidermic dropsy
Showing erythematopapular lesions on the arm and forearm

(Fig. No. 79) which rarely ulcerates. Papillomatous growth is found on the mucous membranes and on tongue



Fig. No. 79
Epedermic dropsy
nodules on back
(Case of
Dr. L. K. Ganguli)

(Fig. No. 80) and at other places. Breathlessness and palpitation are usual accompainment.

Diagnosis (1) Typical pigmentation, erythema blanching and redness on pressure with sarcoid like



Fig No 80
Epidemic dropsy nodule on mucous membrane of lip
(Case of Dr L K Ganguli)

nodules or spider nevus like lesions (2) History of consuming mustard oil (3) Examination of mustard oil for the presence of *argemone oil* (4) Histopathology shows only dilatation of vessels in the dermis

Prognosis Good

Treatment Prophylaxis is to stop consumption of mustard oil Curative is to stop mustard oil and to give the patient high protein diet and vitamins by mouth Liver extract 2 c c I M injection is given daily Ephedra vulgaris dr ½ is given in water thrice daily after food Intravenous inj of 25 p c glucose with 10 p c Calcium and Vitamin C (500 mg) may be given every day Rest in bed in essential No local treatment is necessary for the skin lesions

CHAPTER VIII

BACTERIAL SKIN DISEASES

Skin diseases may be caused by different bacteria producing different types of lesions

The organisms causing disease like

1 Pyogenic infection
2 Tropical ulcer
3 Chancroid
4 Cutaneous anthrax
5 Cutaneous diphtheria
6 Rhinoscleroma
7 Leprosy
8 Cutaneous tuberculosis

Pyogenic Infection: This forms a group of skin infections caused by strepto and staphylococci Common pyogenic infections in the tropical country are

(a) Impetigo contagiosa
(b) Pemphigus Neonatorum
(c) Bochart s impetigo
(d(Furunculosis
(e) Carbuncle
(f) Chronic folliculitis
(g) Summer boils
(h) Dermatitis vegetans
(i) Acrodermatitis perstans and Acrodermatitis tropicalis
(j) Granuloma pyogenicum

Impetigo Contagiosa : Definition—Is an acute contagious superficial, bacterial, skin disease characterized by the formation of vesicles and crust formation.

Etiology : Caused by *hæmolytic group A streptococci* and staphylococci as a sacondary invader but sometimes by both the organisms. Impetigo on the scalp *is due to scratching in* pediculosis and on the body is due to scratching in scabies. Age—No age is exempt. Sex—common in both sexes. Acrodermatitis enteropathica and pemphigus neonatorum affects infants whereas impetigo and granuloma pyogenicum affects the children and all others affect adults in the tropics.

Signs and symptoms : They cause both systemic reaction and damage to the skin. Acute impetigo contagiosa often appears on the face of children (Fig. No. 81) and the infection is due to contact. A vesicle appears with an areola and it bursts and the serum gets dried up and remains on the denuded surface of the skin as a stuck-on crust (Fig. No. 82). Several bullae appear together and is known as **Impetigo bullosum** (Fig. No. 83) There may be systemic reaction also. Bullæ may become generalised also. The generalisation of the impetigo takes place in the new-born infants with systemic reactions when it is called **Impetigo neonatorum** or Pemphigus neonatorum. The bulla in an impetigo ruptures and at the periphery of the denunded surface a *group* of bullae appear causing extension peripherally. This type is called **Impetigo sercinatum** (Fig. No. 84) When the impetigo extends deep down to *the dermis* it is called **Ecthyma. Ethyma-Contagiosum** (Orf) is a virus disease of sheep and goat and is occasionally transmitted to man. *When the impetigo extends*

both peripherally and deeply it is called **Impetigo ulcerativum** Impetigo may produce a circular or

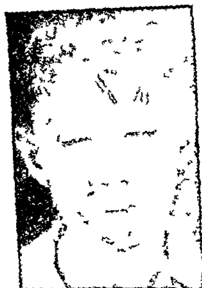


Fig No 81

Impetigo contagiosa

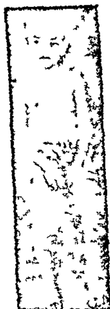


Fig No 82

Impetigo contagiosa

(Lesions are on face, hands and feet)

oval erythematous lesions on the face of a child and become chronic when it is called **Impetigo petyroides** When the impetigenous lesion involves the superficial part of the hair follicle it is called **Bochart's impetigo** (Fig No 80) Affects also the hairy regions involving the whole of the hair follicles when it is called **Folliculitis** May involve any hairy region When chronic and affects beard region it is called **Sycosis barbæ** When affects the nuchal region it is known by the name of **Sycosis nuchæ** and there may

develop keloid which is called **Nuchal keloid** (Fig No 86) When the lesion involves the whole of the hair

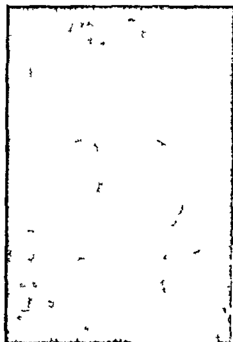


Fig No 83
Impetigo bullosum

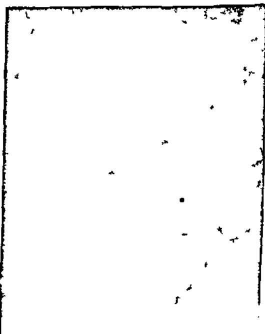


Fig No 84
Impetigo sercinatum

follicle and produces gangrene of the hypodermis it is called **Furunculosis** which occurs most commonly on the

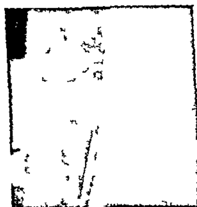


Fig No 85
Bochart's impetigo

neck When several of the hair follicles are involved producing gangrene of a large portion of the hypoderm resulting in systemic reaction it is called **Carbuncle.** Generally affects people in the tropics at 40 years of age and having high blood sugar This condition is associated with grave systemic reaction When suppurates looks

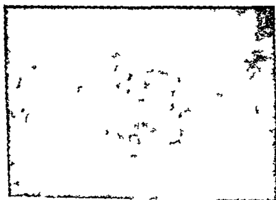


Fig No 86
Nuchal keloid
Back of head

like a honeycomb The infection goes down to the hair follicle and apocrine glands The condition clinically presents a picture of skin coloured nodules Which

suppurates and is known as **Hidradenitis suppurativa.** Axillæ are the common sites. There may be systemic reaction with it. When the infection becomes chronic it affects different apocrine glands and affects the sweat glands including the subcutaneous tissue and fascia. When the infection goes down the spiral ducts and affects the sweat-glands the picture is that of multiple pea-sized to half beetle-nut sized skin coloured lesions which affects mainly the face, neck, shoulders and even the chest and rarely the arms. It is a very painful condition. The lesions change colour from that of the skin to reddish or reddish-brown and suppurate. Commonly affects children in the tropics but sometimes seen in adults also in summer. This condition is called **Summer Boil** or Mangoe boil.

Rarer types of pyogenic lesions are the following:—

The organism produces a low grade localised chronic infection particularly in the intertrigenous areas such as the crural regions, the axillae or even on the skin of the extremities The lesion looks granulomatous, warty or papilomatous with pus oozing out and producing a cauliflower-like growth when it is called **Dermatitis vegetans.** Patients generally suffer from malnutrition and with history of the skin lesion of over 3 to 6 years duration. ***Dermatitis gangrenosum*** is the gangrene of the skin and occurs in association with small pox, typhoid, carbuncle,erysepelas, gas gangrene, in vascular diseases like Buerger's disease, Raynourd's disease arteriosclerosis as in diabetis and also in embolism and thrombosis of arteries. After a trauma in the perionychial region a low-grade bacterial infection causes a very slow forming, superficial, undermined skin lesion which proceeds generally from the thumb or the toe over the palm or sole and is

resistant to all treatment This condition is called **Acrodermatitis perstans** (Hollopeu) or Dermatitis repens (Crocker) which is generally unilateral But in the tropics a similar chronic slowly active, symmetrical lesion either on both palms or both soles occur which may be called **Acrodermatitis tropicalis** In the tropics repeated gastro intestinal infection in children causes stunted growth and even the development of a symmetrical chronic pyogenic moist lesions which travel down from the netal clefts to either side of the medial sides of the thighs involving both the gluteal regions and reach down the knees ankles and the dorsum of feet. Together with this there may be alopecia and nail changes with thrush like lesion on the tongue and buccal mucosa may be present The skin lesion may be present at other places e g circumoral region eye lids and also on the upper extremities This condi ion is seen in undernourished children in the tropics with a history of commencement of the disease at infancy and is called **Acrodermatitis enteropathica** (Fig No 87) A type of rapidly growing pedunculated growth the size of a pea or a beetle nut may occur anywhere on the skin after a microscopic trauma This is due to the entrance of the pyogenic organisms causing a flesh coloured growth This is moist with discharge Injury causes bleeding from its rough surface May occur on face, back, hands and feet This condition is known as **Granuloma Pyogenicum** (Fig Nos 88 & 89) Sometimes multiple nodular chronic painful lesions with necrosis at the centre occur on the face and is called **Staphyloderma chronica** or **Staphyloma** (Fig Nos 90 & 91)

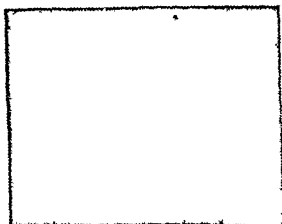
Diagnosis (1) Clinical examination, (2) Smear

suppurates and is known as **Hidradenitis suppurativa.** Axillæ are the common sites. There may be systemic reaction with it. When the infection becomes chronic it affects different apocrine glands and affects the sweat glands including the subcutaneous tissue and fascia. When the infection goes down the spiral ducts and affects the sweat-glands the picture is that of multiple pea-sized to half beetle-nut sized skin coloured lesions which affects mainly the face, neck, shoulders and even the chest and rarely the arms. It is a very painful condition. The lesions change colour from that of the skin to reddish or reddish-brown and suppurate. Commonly affects children in the tropics but sometimes seen in adults also in summer. This condition is called **Summer Boil** or Mangoe boil.

Rarer types of pyogenic lesions are the following :—

The organism produces a low grade localised chronic infection particularly in the intertrigenous areas such as the crural regions, the axillae or even on the skin of the extremities. The lesion looks granulomatous, warty or papilomatous with pus oozing out and producing a cauliflower-like growth when. it is called **Dermatitis vegetans.** Patients generally suffer from malnutrition and with history of the skin lesion of over 3 to 6 years duration. **Dermatitis gangrenosum** is the gangrene of the skin and occurs in association with small pox, typhoid, carbuncle,erysepelas, gas gangrene, in vascular diseases like Buerger's disease, Raynourd's disease arteriosclerosis as in diabetis and also in embolism and thrombosis of arteries. After a trauma in the perionychial region a low-grade bacterial infection *causes a very slow forming, superficial,* undermined skin lesion which proceeds generally from the thumb or the toe over the palm or sole and is

the prese ce of inflam rator sign i the dermis anc lypoderm is seen in sum ner boil In derm titis vegetan



F g No 89
Gra uloma pyoge icum
(Nodule on the back of axilla)



F g No 90
Stapl lo n

tle important changes in tle epiderm is are micro abscesses and pseudol yperplasia In acrodermatitis

examination with Gram's stain will reveal strepto and staphylococci, (3) Culture of the discharge will show the

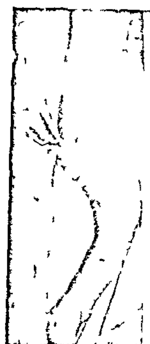


Fig No 87
Acrodermatitis enteropathica



Fig No 88
Granuloma pyogenicum
(Nodular growth on the tip of thumb)

pyogenic organisms (4) Biopsy histopathology will show in impetigo contagiosa an intra epidermal bulla high up in the stratum corneum The lesion reaches up to the dermis The lesion is in the upper part of the hair follicle in Bochart's impetigo Inflammation of the whole of the hair follicle is found in folliculitis whereas in furunculosis there is inflammation of the hair follicle together with gangrene of the hypoderm When there is inflammation in more than one hair-follicle together with gangrenous change of the greater part of the hypoderm it is the picture of carbuncle. There is dilatation of the sweat gland together with

pemphigus vegetans, drug rash (bromoderma) and tuberculosis verucosum cutis, (4) Acrodermatitis tropicalis of palm and sole has to be differentiated from psoriasis palm and sole, (5) Acrodermatitis entero pathica from psoriasis, (6) Granuloma pyogenicum from haemangioma and subungual melanoma

Prognosis Is good in every case when properly diagnosed and systematically treated

Is good in granuloma pyogenicum with excision

Treatment Prophylaxis—In the tropics prophylaxis is very difficult With the raising of the standard of living and better knowledge of scientific diet and hygiene various skin diseases which are due to infection and trauma are not likely to occur so much

Curative—Impetigo is treated by removing the bullae roof under aseptic condition followed by washing with boiled warm water and a bland soap Then it is dressed every hour with the lotio No 1

No 1	Argentii Nitras	gr 5
	Aqua Distil	oz 1

Lotio for external use Supply oz20 in a coloured phial Linen soaked in this lotion is applied every hour for a day or two when the lesions get dried up When dry lotion No 2 is painted once daily

No 2	Gentain violet	gr 5
	Aqua Distil	oz 1
	Lotio for external use	

tropicalis there is acanthosis of the stratum mucosum with papillomatosis and there may be found micro-



Fig. No. 91
Histopathology of Staphyloma

abscesses. Granuloma pyogenicum shows dilatation and proliferation of vessels in the dermis together with infiltration with polymorphonuclear cells and mast cells

Differential Diagnosis: (1) Impetigo is *to* be differentiated from herpes *zoster, chicken pox, urticaria* bullosum, dermatitis herpetiformis and epidermolysis bullosum, (2) Impetigo peteroides has to be *differentiated* from seborrhoic dermatitis and contact *dermatitis*, (3) Dermatitis vegetans has to be distinguished from

pemphigus vegetans, drug rash (bromoderma) and tuberculosis verrucosum cutis, (4) Acrodermatitis tropicalis of palm and sole has to be differentiated from psoriasis palm and sole (5) Acrodermatitis entero pathica from psoriasis, (6) Granuloma pyogenicum from haemangioma and subungual melanoma

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No 2	Gentain violet	gr 5
	Aqua Distil	oz 1
	Lotio for external use	

When it gets dried up the skin is kept bandaged up with an ointment No 3 or Zichthol (Gantosan)

No 3	Ichthyol	gr 5
	Vaseline alba	oz 1

Ft Ung for external use At night dressed only with ointment No 4

No 4	Hydrarg Ammon	gr 10
	Pulv amylum	dr 1
	Zinc oxide	dr 1
	Vaseline alba	oz 1

Ft Ung for external use

No 5	Ichthyol	dr 4
	Glycerine pure	dr 4
	Ft paint for external use	

When there is systemic manifestation of toxicity like fever sulphadiazine or Elkosin is given for 4 days orally 0 5 g at 6 hours for adult but rarely Penicillin crystallin 'G is injected in 0 2 Mega Unit dose twice daily for 5 days only In impetigo pteroides only ointment No 4 is applied In folliculitis in the acute stage hot Condy s bath followed by shaving of the part is essential and then painting twice daily of the lotion (No 2) followed by application of an ointment (No 3) and another ointment (No 4) only at night When chronic crude liver extract is injected biweekly in dose of 2 c c I M Carbuncle is treated by hot fomentation 4 times daily with injection intramuscularly twice daily of 0 2 Mega unit of Crystalline penicillin 'G' for 5 to 7 days When sloughing occurs S S Magsulph dressing is helpful When slough clears

away dressing is done with an ointment (No 4). Hydradinitis suppurativa is treated with hot fomentation followed by application of a paint (No 5) locally applied 4 to 6 times daily together with Sulpha therapy or Penicillin injections Sometimes requires excision of the glands Summer boils should be treated by hot fomentation and application of a paint (No 5) together with sulpha drug Rarely needs operation.

Dermatitis vegetans should be treated internally by high vitamin C and locally with Condy's bath and a lotion (No 2) is painted followed by the application of an ointment (No 3) and after 2 to 4 days treated with another ointment application (No 4) Antibiotic may be used orally Acrodermatitis tropicalis requires locally an ointment (No 6)

No 6	Acid salicylic	gr	10
	Zinc oxide	dr	½
	Liq Picis carb det	m	10
	Vaseline alba	oz	1
	Ung for external use		

Later on painting the part daily with a paint (No 7) and dusting over with a powder (No 8) is helpful

No. 7 10 p c Crude Coal tar in acetone for external use

No 8	Pulv zinc oxide	dr	3
	Pulv acid salicylic	dr.	1
	Pulv Amylum	ad oz	1

In acrodermatitis enteropathica orally is given diodoquin (diodohydroxyquinoline) 100 mg. four times daily

for a week, 100 mg thrice daily for a week, 100 mg. twice daily for a week and ultimately 100 mg. once daily for another week before stopping the drug Locally treated with painting of a lotion (No. 2) followed by application of an ointment (No. 3) during the day and only the application of another ointment (No. 4) is advocated at night. High protein diet and multivitamin (Panlyn of Calcutta Chemical) are advocated.

In granuloma pyogenicum removal of the growth by excision followed by the application at the base of Tn. Iodine or Silver nitrate stick is essential. X-ray therapy is sometimes advocated.

TROPICAL ULCER

Definition: Is a chronic ulcer of the leg occuring in tropics due to fusiform bacillus and spirocheta of Vincent.

Etiology. Commonly seen during the monsoon all over the tropics. Age—any age. Sex - Common in males. Trauma is a predisposing factor but malnutrition particularly of protein and vitamin C is the main etiological basis in the causation of tropical ulcer Various organisms are found such as *staphylococci, streptococci* but the predominating organisms are fusiform bacillus and spirocheta vincenti

Signs and symptoms After some trauma starts as a red papule on which a bleb of the size of a pea is formed. The bulla ruptures leaving a small ulcer. The ulcer is round with an undermined irregular edge. The base of the ulcer may be covered by a crust or a pseudo-membrane The ulcer is usually a single

but multiple ulcers may be seen. Site is commonly the leg and other exposed parts like the dorsum of the hands. Ulcers may be superficial but sometimes it may extend deeply destroying the underlying structures like muscles, tendons and even affects the periosteum and the bone. It is a tender and painful sore. There may be systemic reaction with it.

Diagnosis · (1) Indolent solitary ulcer on the leg in a tropical country, (2) Ulcer is tender and is covered with a pseudo membrane and there is a red hallow, (3) Smear examination from the ulcer will show fusiform bacilli and spirocheta vincenti, (4) Vitamin C in blood is subnormal, (5) Serum protein shows low total protein with low albumin, (6) Biopsy—histopathology shows edema in the dermis with many fusiform bacilli

Differential diagnosis (1) Diphtheretic ulcer, (2) Varicose eczema, (3) Syphilitic ulcer, (4) Scabies.

Prognosis Good with rest and proper treatment,

Treatment Prophylactic—patient should avoid trauma Regular use of foot wears is helpful. Nutrition must be improved Vitamins, particular A and C, should be taken in prophylactic dose.

Curation—(1) Locally warm Condy's soaks 4 times daily followed by the application of Mag sulph pastes for a day or two then an ointment (No 1) containing

No. 1.	Acreflavin	gr. v.
	Oil Morrhœ ...	oz. 1

Antibiotics may also be used such as aureomycin one capsule (250 mg) every 6 hours with Vitamin B complex orally or combiotic may be injected once daily 7 to 10 days Same treatment is given to the bulbo when develops Ducrey bacillus vaccine may also be injected

CUTANEOUS ANTHRAX

Definition is an acute specific skin disease characterized by a pustule on a wide erythematous base accompanied with systemic reaction and is caused by Bacillus anthracis

Etiology Anthrax although is rare these days but once or twice a year a patient is seen in a large hospital Anthrax is not infrequently seen in the industrial areas dealing with raw hide and fur Commonly seen in those who handle hide and fur Cases of anthrax have occasionally been reported by using infected shaving brushes

Signs and symptoms Earliest lesion is a red elevated area of about $\frac{1}{8}$ inch diameter This red area becomes larger and a central black area appears called *eschar* Surrounding this black area are tiny vesicles and this is surrounded by a red œdematous area and the whole lesion is called the **'Malignant Pustule'** *Together with the development of this lesion the part becomes* tense and tender. Constitutional symptoms develop consisting of high temperature, headache, toxicity and albuminuria with prostration is the picture in acute cases Lymphadenitis is present which often suppurates Chronic cases commonly show enlargement of regional lymph glands Rarely secondary lesions due to autoinoculation appear

Diagnosis (1) Clinical picture (2) Smear from the vesicle or pustule shows the presence of B anthracis ; Which are rod shaped and squre shaped, gram positive bacillus (3) Culture shows Bacillus anthracis (4) Blood examination for total count will show leucocytosis and high neutrophil count, (5) Histopathology—shows destruction of the epidermis and spongiosis Rarely an intraepidermal bulla is found There is oedema in the dermis and W B C and R B C are found present in it Anthrax bacilli are also seen

Differential Diagnosis (1) Impetigo (2) Drug rash, (3) Erysipelas

Prognosis Is good with modern treatment

Treatment Prophylactic—sterilisation of shaving brush is essential In trade those who handle hide and fur should use gloves and aprons Prophylactic inoculation is valuable with Bacillus anthracis vaccine

In the management of a case of anthrax the case should immediately be hospitalized in special infectious disease ward with specially trained nursing staff

Patients should have rest in bed with plenty of fluid to drink and alkali to flush the kidneys Sulphadiazine or sulphapyridine in dose of 0 5 to 1 0 gm orally is given every 4 hours for a week with the examination of blood on every alternate days for agranulocytosis Together with this treatment Sclavo s serum 3 c c I V is given when available otherwise as a routine (1) Orally sulphapyridine 0 5 gram 4 hourly for 7 days (2) Combiotic injections intramuscularly for seven days is helpful, (3) Aureomycin (250 mg) capsule to be given

every 6 hours for two days then one every 6 hours for 8 days with vitamin B complex

Locally 1 p c Ung Ichthyol dressing is applied

CUTANEOUS DIPHTHERIA

Definition Is an ulcerated skin lesion caused by Cornybacterium diphtheriæ

Etiology All ages and both sexes may be affected Caused by C diphtheriæ Is found in 0 02 p c in the tropics

Signs and symptoms The lesions are commonly found on the inferior extremities The lesions may be eczematous or cellulitic or as chronic indolent ulcers There may be associated paralysis of the limb

Diagnosis (1) Indolent ulcers on legs with or without paralysis (2) Biopsy—histopathology shows the inflammatory reaction with cornybacterium diphtheriæ (3) Smear from the ulcer shows with special stain C diphtheriæ (4) Culture of the pus from ulcerated growths for C diphtheriæ is positive

Prognosis Good if early diagnosed but paralysis may develop.

Treatment Prophylaxis in suspected cases 20,000 i u antidiphtheretic serum is injected Curative 50,000 i u should be injected immediately and should be repeated daily for 3 days in dose of 25 000 units Penicillin crystalline 'G' should be injected I M daily for 3 days in dose of 5 lacs

RHINOSCLEROMA

Etiology Rhinoscleroma is a chronic granulomatous skin disease of the nose caused by a gram negative organism called Bacteria rhinoscleromatosis

Etiology Is commonly found in central and upper India Seen commonly amongst young adults and found in both sexes Incidence is about 0 05 in the tropics

Signs and symptoms Starts as a red nodule on the side of the nose which grows very slowly and invades the nares and ulcerates (Fig No 92) The foul nasal discharge is the early symptom but later on obstruction develops due to growths in the nose, nasopharynx and also in the larynx



Fig No 92
Rhinoscleroma

Diagnosis (1) Clinical features, (2) Biopsy—histopathology shows fairly large number of vacuolated histiocytes called 'Mikulicz cell' containing

every 6 hours for two days then one every 6 hours for 5 days with vitamin B complex

Locally 1 p c Ung Ichthyol dressing is applied

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LEPROSY

The general practitioner should consider leprosy like any other bacterial disease and should help to educate the public accordingly The taboo of leprosy is the cause of its spread in the East Leprosy is becoming a menace in India The clinician of today is equipped with the modern chemotherapeutic drugs to fight the scourge of leprosy About 2 per cent of all skin cases seen in India are cases of leprosy now

Etiology Leprosy is caused by an acid fast bacillus called Bacillus lepræ or Hansen's bacillus Hansen discovered the organism in 1874

Incubation period is not known May be from several months to years It is a low grade contagious disease in all the stages but at a certain stage of the disease it is infectious but at others it is not Nutrition plays a great part both in the spread of the disease and also in its effect on the system It is not a familial disease and a child separated after birth from a leprous mother may escape infection Upto the age of 3 years the possibility of getting the infection is maximum which gradually decreases with the growth of age People have also been found to get the infection at a late age probably by mucous membrane contact

Signs and Symtoms Classification (1) **Lepromatous type**—nodules are found all over the body On the face nodules (Fig No 94) appear which when coalesce gives a

bacilli with plasma cells, leucocytes, lymphocytes and histiocytes (Fig No. 93).



Fig No. 93
Histopathology of rhinoscleroma

Differential Diagonosis · (1) Syphilis, (2) Leprosy,

Prognosis: Is fair.

Treatment: Streptomycin is injected in gram *1* dose for 21 days with PAS 0·5 gm. tablet—2 tablets 6 times daily after food. Locally 10 p.c. PAS ointment.

the body may be affected (Fig Nos 97 & 98) Lepra fever may also develop Nodules may appear on the



Fig No 96
Trophic ulcer
(On the planter surface of the great toe in a leper)

cornea destroying the eyes Lymphangitis may occur. Claw-hand may develop and is called **"leper claw"**.



Fig No. 97
Leprosy on left cheek
(Intermediate type)

lion-like face called **leonine facis.** The ears and nose look larger in size. Edema appears and fingers and toes

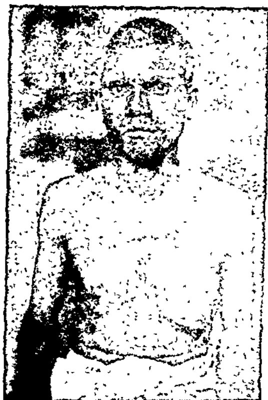


Fig No. 94
Lepromatous leprosy showing unilatral gynecomastica in a male aged 35 years.
(Case of Captain S N. Roy)

look tense and separated. Ulceration of fingers (Fig. No. 95) and toes and ulcers also appear under the soles when



Fig. No. 95
Leprosy
(Tuberculoid type)

called **trophic ulcers** (Fig. No. 96). Skin of any part of

the presenting sign Thickening of the cutaneous nerve (Fig No 100) supplying the area is often the earliest sign found There may be anaesthesia or hyperesthesia Gynecomastica (Fig No 94) may develop in males which is unilateral or rarely bilateral

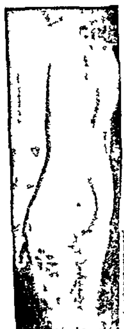


Fig No 99
Leprosy
(Tuberculoid type)

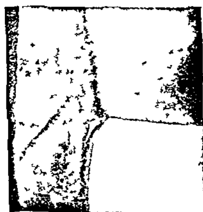


Fig No 100
Tuberculoid leprosy
(Thicked nerves behind left elbow)

The latest Indian classification of leprosy of 1955

(1) Lepromatous (L)
(2) Tuberculoid (T)
(3) Maculoanaesthetic (MA)
(4) Polyneuritic (P)
(5) Border line (B)
(6) Intermediate (I)

Thickening and abscess of nerves are usually seen. Loss of hair, depigmention or pigmentation of the skin may be associated. Epistaxis is a common symptom.

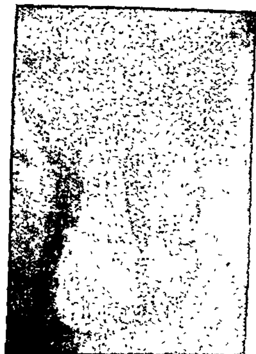


Fig No. 98
Leprosy of the Scrotum

(2) When the disease becomes chronic it is called the **Intermediate type.** This type may develop also when a tuberculoid type of leprosy develops into the lepromatous type. Lesions are erythemato-papular. Here *the acute symptoms and signs of lepromatous stage are absent but merely exaggerated signs and symptoms than* the tuberculoid type are seen. This is also somewhat infectious.

(3) **Tuberculoid type** *of leprosy is not infectious.* It presents *varied clinical picture.* Macular depigmented, hyperpigmented, erythematous, anhidrotic areas may be the only sign (Fig. No. 99). *Macular depigmented patch* with circumferentially hyperpigmented area also may be

the presenting sign Thickening of the cutaneous nerve (Fig No 100) supplying the area is often the earliest sign found There may be anaesthesia or hyperesthesia Gynecomastica (Fig No 94) may develop in males which is unilateral or rarely bilateral

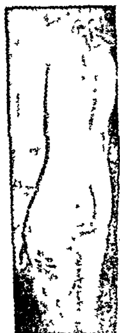


Fig No 99
Leprosy
(Tuberculoid type)



Fig No 100
Tuberculoid leprosy
(Thickened nerves behind left elbow)

The latest Indian classification of leprosy of 1955

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(2) Tuberculoid (T)
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(4) Polyneuritic (P)
(5) Border line (B)
(6) Intermediate (I)

Diagnosis: (1) Clinical *examination for thickening* of skin and nerves as well as depigmented and hyperpigmented areas of skin.

(2) Pin prick and cotton wool touch test. Sometimes instead of anaesthesia the *patches may* be hyperaesthetic. Epicritic sensibility is first lost. Temperature sense may disappear first hence the importance of testing with test tubes filled with cold and warm water In early tuberculoid type the loss of temperature sensation may be the only sign and *in the fully developed* tuberculoid type as *well* as in all the other types the sense of pin-prick and cotton-wool touch are lost. Deep sensibility and gross pain *sensations are also lost in the Intermediate* and Lepromatous types of leprosy

(3) Skin scraping smear examination for Bacillus lepræ and staining with Zeil Neelsen stain for acid fast bacilli.

(4) Nasal smear examination for acid fast staining.

(5) Urine examination routine

6) E. S. R of fasting blood higher reading in leprosy-moderately high (40 mm) in the intermediate and highest (70 mm or more in lapromatous type)

(7) Blood W R Pseudo positive

(8) *Blood examination for total* and differential count, Hæmoglobin p c and parasite

(9) Biopsy of the lesion and histopathological examination will show atrophy of the epidermis with Bacillus lepræ in the dermis is commonly seen in the

Lepromatous stage less commonly in the Intermediat stage and rarely in the Tuberculoid stage of leprosy Giant cells are seen in the Tuberculoid state Perineural infiltrat is also a feature of leprosy Destruction of the cutaneou nerve is commonly found in the tuberculoid type c leprosy Tuberculoma is seen in tuberculoid leprosy (Fig No 101) Perivascular infiltration of histiocytes, lymph



Fig No 101
Section of Tuberculo
Leprosy showing tuberculoma
(Case of
Major B Chukarbutt

cytes and plasma cells in the upper part of dermis characterstic and in the lepromatous stage the prese of large vacuolated histiocyte is called Lepra cell' Globi body

Differential Diagnosis (1) Post Kala Azar derr leishmaniasis (2) Syphilitic cutis (3) Vitili (4) Ringworm (5) Tuberculosis cutis (6) Sarcoido (7) Pemphigus

Prognosis It is good with modern treatment

Treatment Improvement of the general nu tional stage of the patient is essential Patient should

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(9) Biopsy of the lesion and histopathological examination will show atrophy of the epidermis with Bacillus lepræ in the dermis is commonly seen in the

2 to 5 years as need be The treatement is a prolonged one and the co operation of the patient is absolutely essential in the cure of the disease

Liver extract injection weekly are good adjuvant treatment during the one day rest period while on sulphone therapy

Isoniazide in dose of 200 mg by mouth in divided doses is given alone or with D D S for 6 to 12 months

Lepromatons reaction—Is characterized by exacerbation of the skin lesions and even there are new formation of lepra nodules in the eyes all over the 'skin and viscera together with systemic reactions In this stage sulphone *is useless* *Streptomycin* or Dihydrostreptomycin in one gram dose is injected I M for 10 days

Sodium salt of chaulmoogra oil is injected intradermally and also I M weekly

Rehabilitation of the leprosy patients in the leprosorium is advocated Eradication of the disease is not difficult from a country but this is a problem of social medicine

TUBERCULOSIS

Tuberculous disease of the skin is becoming quite common in the tropics In one of the big *cities in India*, it has been estimated that out of all skin cases attending about 1 5 p c are tuberculous The approximate average number of tuberculous skin cases in India has been estimated to be about 2 0 *p c Either the* skin consciousness amongst the public or the *increase* in the incidence of this disease is responsible for this high percentage

given high protein diet with plenty of green vegetables and milk. Liver diet is preferable.

Medicinal: Sulphone therapy If the blood examination shows no agranulocytosis and Hb p.c. is above 70 it should be started and can be given in all stages of leprosy. The blood examination should be repeated weekly as well as the examination of urine.

Novophone Y (Bengal Chemical), Sulphone (Cilag), Siocarbazone (Albert David), Sulphetrone tablet (B. W.) or D.A.D.P.S (I. C. I.) tablets are given as follows :—

Started with ½ tablet once daily and is give for 6 days a week and no tablet on the 7th day. ½ tablet twice daily for 6 days a week and repeat for 5 weeks. Patients who do not tolerate sulphone orally may be given intramuscular injection of 50 p.c. sulphetrone or Novotrone solution ½ to 1 c. c. weekly in first week then biweekly for 12 weeks. There are various other sulphone preparations available in the market. Diazone (Abbott) may be given as one tablet daily for a week then twice daily for 5 weeks followed by rest for a month. Repeat sulphones for 2 years with iron.

Patient should have rest for 4 weeks during which the examinations of E.S.R., skin snip smear for B. Hansen and even biopsy should be repeated and during this time injections intradermaly and intramuscularly of Hydnocreol weekly are given or Streptomycin or Dihydrostreptomycin is injected I. M. daily for 10 days and repeating for 10 days after an interval of a day with PAS (4 grams T.D.).

After the examinations when results are negative and absence of bacillus the treatment should be continued for

Age-Generally seen in the young age Children suffer mostly. 80% of cases are seen under the age of 20 years. But accidental inoculation with Koch's bacillus may occur at any age particularly in a family where there is present a case of open pulmonary tuberculosis. Inoculation following vaccination or piercing of ear lobules of girls for putting on ornaments or after tattooing skin tuberculosis may develop at any age. "Postmortem wart" on the fingers of doctors who are doing autopsy or are handling dead bodies in the anatomy hall develop warty tuberculous skin lesions on the fingers. Warty lesions are also found on the sole after a thorn prick (Fig. No 102).



Fig. No 102
Lupus verrucosum plantaris

is a problem to the specialists in skin disease in India at present A better dermatological knowledge amongst the general practitioners in the tropics is certainly helping to *bring to light a larger number* of cases of *skin tuberculosis* and is also helping in the treatment with the modern drugs

Definition Is a chronic granuloma of the skin due to Bacillus tuberculosis

Etiology Tuberculous disease of the skin is caused by Mycobacterium tuberculosis Koch described four different types of bacilli human bovine avian and piscine In India the human type is responsible for cent per cent cases of *skin tuberculosis*

Mycobacterium tuberculosis *is a* rod shaped, non motile, acid fast organism (A F B)

Tuberculosis is not a hereditary disease but children *born of tuberculous parents have susceptibility* for this disease

Mode of infection

(1) Direct through the broken skin

(2) Direct contact with an infected tuberculous gland or a sinus

(3) Auto infection such as from an open lesion either in the lungs or anywhere in the body,

(4) Hæmatogenous *infection*,

(5) By extension peripherally,

Sex Females are commonly effected In the tropics both the sexes are equally affected

discharging sinus Koch s bacilli may be cultured if pus is inoculated in a guinea pig in the laboratory and the ulcer



Fig No 103
Primary Tuberculous complex
(Case of Dr B N Banerji)

in the finger or toe shows clinical tuberculous granuloma which on pressing with a glass slide (Diascopy) shows yellowish granules (apple jelly nodules) Tuberculin te t (Manto ix) is negative in the early stage and only becomes positive after 8 weeks There is no other tubercular focus anywhere in the body The skiagram of the chest may be negative in primary tuberculous complex and the Mantoux test positive Healing is by fibrous tissue formation and calcification with very little scar formation

(2) **Tuberculosis Verrucosum Cutis**—This is quite common and is often seen in hospital and in special practice Patients may be of any age but particularly common amongst children Hands and feet are the common sites Lesions are warty, indurated, dry and found particularly on the palm sole (Fig No 102) and knuckles of fingers There is always present a history of injury or pin prick The Koch s bacillus enters through the

Pathology-After the Mycobacterium tuberculosis enters the tissues a non specific inflammatory reaction starts The introduction of living or dead tubercle bacilli or their disintegration produces a granulomatous lesion which shows histologically giant cells, epitheloid cells and lymphocytes Subsequently inocculation of the body with tubercle bacilli will produce a granuloma The formation of granuloma depends on the virulence of the organism and on the resistance of the tissues. Allergic reaction to the Koch's bacilli and its toxin may take a great variety of forms The essential features of these reactions are that they are benign eruptions, symmetrical and often peripheral in distribution, showing a tuberculous histology without demonstrable presence of the organism and often occurring in tuberculous subjects These benign eruptions are called *tuberculids* Tuberculids are of various types Damage to the skin with tubercule bacillus is not so severe in the tropics and the incidence of cutaneous tuberculosis is much less on the pigmented skin in the tropical country

Classification Tuberculosis of the skin is mainly of two different types such as (A) Localized and (B) Hæmatogenous (A) Localized type may further be subdivided as follows

(1) **Tuberculous chancre**—which is the **"primary tuberculous complex"** It consists of two parts such as a primary tuberculous sore on the skin and a suppuration of the regional lymph glands (Fig No 103) It has been observed in children and in adults in the tropics There may be an injury on the finger or toe followed some times after by lymphadenitis The ulcer becomes indolent and the lymphadenitis suppurates and breaks leaving a

nose and the like Any other part of the body may be



Fig No 104
Lupus Vulgaris
(Lesions over the Knee joint and on in thigh look verucose)

effected (Fig No 106) with lupus vulgaris giving the typical diascopic appearance of apple jelly In debilitated subject the skin lesions break down (Fig No 107) and ulcer is formed Lupus vulgaris of soft parts like mouth and nose produces deformities and may take different forms such as pastular serpiginous and vegitative Lupus of the limbs (Fig No 108) may result in solid edema Lupus vulgaris may affect other parts of the body also Tuberculin test is always positive in lupus vulgaris

(5) **Scrofuloderma**—is due to the local effect of the tubercle bacilli on the skin from some underlying tuberculous glands bones and joints Common sites

breach in the skin. The *infection is always produced* by direct intracutaneous entrance of the bacillus. Tuberculin reaction is always found positive. Sometimes seen amongst medical practitioners and nurses. The regional lymphadinitis may be present.

(3) **Tuberculosis Cutis Orificialis**—*the condition is* found in patients with pulmonary tuberculosis. Common sites of the ulcers are the muco-cutaneous junction of lips and the tip of the tongue. Indolent shallow ulcers may be found on the tip of the tongue but may also be present on the lips, anal region and on the glans penis. Lesions may be nodular, ulcerated or papillomatous in types.

(4) **Lupus Vulgaris**—is the commonest tuberculous skin disease and is easily diagnosed even by the busiest general practioner. Inoculation lupus vulgaris is not uncommon Lupus vulgaris can develop in a person on the site of B.C.G. vaccination 2 years after. Tuberculous nodules develop and coalace to from the lesion of lupus vulgaris. Some of the lesions become verrucous (Fig No. 104) and remain small in size. Any part the body may be affected but the commonest site is the face (Fig. No. 105). In the face the disease starts as a soft, small, yellowish nodule near the nose and spreads very slowly. Sometimes it gets generalised and is called "**Lupus disseminatus**". If a glass slide is pressed on the skin (diascopy) yellow nodules are seen which is called apple-jelly nodule Soft shiny nodules spread like a sheet *peripherally and sometimes shows scales* also. Lupus vulgaris may affect the whole face giving a dreadful appearance with drawing down of one corner of the upper or lown lip, ectropion, pinching of the

affected due to the dissemination of the organisms of pulmonary tuberculosis after measles and whooping



Fig No 166
Lupus vulgaris on the upper arm

cough The skin lesions are like pin head to half lentil in size Sometimes rash may be haemorrhagic also Bacilli are found all over the body Tuberculin test is negative

(2) **Tuberculosis miliaria disseminatus faciei**

are the neck (Fig. No. 109) and joints. Commonly seen in young people. The disease starts as a hard subcutaneous



Fig. No. 105
Lupus vulgaris of face.

nodule which gets adherent to the overlying skin. In time the skin ulcerates and an indolent ulcer or a fistula results. Ulcers may be of different shapes but the edge is undermined and the base is fixed. Cicatrix results on healing. Scrofuloderma is in reality the cold abscess of the skin. Tuberculin test is positive.

(B) Haematogenous type of the tuberculous disease of the skin often starts from some internal tuberculous focus and is subdivided as follows

(1) **Acute miliary tuberculosis of the skin** is rare in the tropics. Children are particularly

may occur on the face when it is called **Acnitis.** Eruptions may occur on the hands when it is



Fig No. 108
Lupus vulgaris of forearm.

called **Follicles.** Lesions are found *on the trunk* and lower limbs also (Fig. No, 110). Lesions start on the subcutaneous tissue and becomes pustular. Central necrosis appears. Lesions heal with a punched-out sca

face is the only part of the body affected. Lesions are



Fig. No 107
Lupus vulgaris

papular and brownish in colour Papules are like half-lentil in size. The lesions come out in crops and may disappear spontaneously leaving pitted scars. Diascopy shows apple-jelly characteristic of the lesion. Tuberculin test is not helpful as it sometime gives positive and at others negative reactions.

(3) **Rosacea-like tuberculid**—these are reddish papular lesions half-lentil in size, appearing in large number and are distributed symmetrically over the cheeks and also on the forehead Lesions do not undergo necrosis. Apple-jelly characteristic is present on diascopy. Commonly seen in women. Takes a long time to heal. Tuberculin reaction is positive.

(4) **Papulo-necrotic tuberculid** occurs at any age but is commonly seen in adults. Affects both sexes. It is a quite common skin condition in the tropics. Eruptions

is commonly seen Lesions are painless and never itch Lesions are grouped and often appears in crops Tuberculin reaction is positive

(5) **Lichen scrofulosorum** is characterized by groups of pinhead sized papules without itching on the trunk and the extremities Involution takes place but relapse is quite common Tuberculin reaction is positive

(6) **Erythema nodosum** is charactrized by pea sized and little bigger sized dusky red nodules on lower extremities below the knees (Fig No 111) These are painful and non ulcerating in nature Tuberculin test may be negative



Fig No 111
Erythema Nodosum
(Case of
Dr B N Banerji)

(7) **Erythema indurata**—is a chronic recurring skin lesion of the legs of young people Commonly seen in girls just over teens and is seldom found in boys Distribution is bilateral and is situated on the calf muscles Subcutaneous erythematous nodules appear which coalesce and becomes a dusky red cutaneous nodule of about ½ to 1 inch in diameter These nodules may

and pigmentation Crusts form under which ulceration appears which heals with a scar Spontaneous healing



Fig No 109
Surofulodermia
Chin Strap type of
Lupus vulgaris



Fig No 110
Papulo necrotic tuberculid
(Case of Dr B N Banerjee)

stage of the disease cutaneous tuberculosis has no typical picture except that there is a nonspecific infiltration with polymorphonuclear leucocytes, hystiocytes and tubercle bacilli may be found, (b) In the advanced stage the epidermis shows no change but in the dermis there may be necrosis surrounded by epitheloid cells and giant cells (Fig. No 112), (4) Guinea pig inoculation is done in the laboratories



Fig No 112
Histopathology of Lupus Vulgaris showing giant cells and epitheloid cells

Differential Diagnosis (1) From dermal Leishmaniasis (a) Tropical residence, (b) Skin snip smear for Leishman Donovan bodies is negative, (c) Biopsy examination does not show Leishman Donovan bodies in the dermis (d) No history of K A and having had no antimony injection, (2) From cutaneous syphilis—particularly mistaken with nodulo cutaneous syphilide (a) Negative Kahn test, (b) Skiagram of bones does not show syphilitic changes, (c) Biopsy does not show

involute or ulcerate with necrosis of the subcutaneous tissue and the underlying tissue. It is tender and painful. Course is very chronic. Tuberculin reaction is positive.

Sign and symptoms : Site is characteristic with a particular type of skin tuberculosis. Lupus vulgaris commonly occurs on the face but may occur at other place as well. Tuberculosis miliaria disseminata facie, Rosacea-like tuberculid and acnitis occur on the face whereas papulo-necrotic tuberculid occurs on the trunk and limbs. Erythema induratum occurs on the calfs of both the legs Lichen scrofulosorum occurs on the trunk and on the extermities and verucosum cutis occurs on fingers, toes, knees, elbows and on planter surface of feet. Spread of the disease is very slow. Tuberculous nodule formation is the primary skin lesion which when coalesce gives rise to different forms. On pressure over the nodules with a glass (diascopy) yellow coloured granules (apple-jelly) are seen. Tuberculous lesions heal by cicatrix formation. It spreads centrifugally and heals at the centre by cicatrization. Tuberculous lesions are very indolent and take a long time to heal.

Complication : (1) Eczema, (2) Impetigo, (3) Solid Oedema, (4) Cutaneous horn, (5) Carcinoma.

Diagnosis : (1) Clinical Examination, (2) Tuberculin Reaction test known as Montoux Test—intradermal injection is given with 0 1 c.c. of old tuberculin in dilution of 1 in 10,000 or 1,000 or 100 on one arm and with sterile distilled water on the other arm. If positive, edematous flush appears within 48 hours. Sometimes there is a central necrosis also. This test is very helpful in the diagnosis of tuberculosis cutis, (3) Histopathology—(a) in the early

weekly or biweekly for a period of 6 to 9 months For a child the dose is reduced according to the age As a routine streptomycin daily or dihydrostreptomycin injections intramuscularly 1 gm per day for three weeks together with PAS 3 g. 4 times a day by mouth for 2 months and then following up the patient with calciferol 50,000 i u twice daily by mouth for 6 months have been found useful in the tropics

Vitamin D2 is a useful drug but shortly after starting the treatment there is congestion in the skin lesions and also in any internal tuberculous focus This active reaction of the tuberculous focus may be fatal to the patient Impairment of the renal function may occur This Vitamin mobilises phosphorous and calcium. Metastatic calcification of soft tissues may occur Improvement in lupus vulgaris with injections in the lesions of high potency Vitamin D2 at intervals of 2 to 3 weeks particularly in localized refractory lesions has been found helpful

Investigations necessary before calciferol therapy are (I) Skiagram of the chest to eliminate pulmonary tuberculosis, (II) Serum calcium and serum phosphorous level estimation, (III) Examination of cardiovascular system, (IV) Blood urea estimation

Calciferol intoxication durig treatment should be watched which manifest as follows —

(1) General weakness, fatigue, loss of weight, (2) Gastrointestinal nausea and vomiting, (3) Neuropsychiatric headache, parasthesia, vertigo (4) Hæmatological normocytic and hypochromic anæmia, (5) Urological-

the presence of numerous plasma cells in the dermis and does not show thickening of the arteries, (3) From Leprosy (a) Impairment of sensation and nerve thickening are absent, (b) skin snip smear and nasal smear for B Hansen negative (c) Biopsy will not show B Hansen in the dermis No lepra cell is formed (5) From Lupus erythematosus (a) Distribution it not typical No atrophy of skin with peripheral pigmentation and scaling are seen, (b) Biopsy gives no characteristic picture of L E, (6) From Epithelioma histopathologically no characteristic of malignancy are seen, (7) From Acne Rosacea—histopathology is helpful, (8) Bromoderma and Iododerma histopathology is helpful, (9) Sarcoidosis—by histological examination and skiagram examination of chest hands and feet, (10) Erythema Indurata from syphilitic gumma

Prognosis Is good with modern treatment in all cases of tuberculous disease of skin with few exceptions

Treatment (A) General treatment amounts to (1) Healthy surrounding with plenty of sunshine and fresh air (2) Good food rich in fat and vitamins and protein are also very essential Table salt is restricted

(B) Local treatment consists of (1) 50 p c *P A S.* ointment as a dressing (2) Isonicotinic Acid Hydrazide ointment (1 in 100 INH) has been found to be effective (3) Ultra violet light exposure weekly is helpful

(C) Specific treatment consists of (1) Calciferol or Vitamin D2—this may be given orally or by injections in doses of 100 000 I U daily by mouth for a young adult otherwise healthy 600,000 I U injection intramuscularly

Calciferol may be used thereafter for a further period of 3 months If there is ulceration present 50 p c P A S ointment locally is indicated When sinus is present 20 p c P A S solution is introduced every day

Radiotherapy is also helpful in tubercular lymphadenitis in association with scrofuloderma of the neck or groin

SARCOID

Definition is a chronic granulomatous skin disease characterized by symmetrical plaque like nodular brownish skin lesions on the face and extensor surfaces of the limbs but bones and lungs, liver and lymph glands may be affected also

Etiology Cause is not known Bacillus tuberculosis is said to be the causative organism It is also thought to be a type of lymphoblastoma Is found 0 1 p c in tropical practice

Age No age is exempt Sex in both sexes

Types (1) Benign sarcoid of Boeck

(2) Lupus pernio of Besnier

(3) Subcutaneous sarcoid of Darier Roussy

Signs and symptoms Boeck s sarboid is red or brown half pea sized nodules developing on the face and the upper extremities which are distributed symmatrically Lupus purnio of Besnier is manifested by infiltrated brownish plaques and is symmetrically arranged Subcutaneous type of Darier Roussy type is characterized by the slow

albumin R B C and cast in urine Slightly elevated alkaline phosphatase level and progressive nitrogen retention, (6) X ray shows particularly of soft tissue calcification, (7) Ophthalmological examination band Keratitis (8) Uræmia, (9) Hypercalcemia

Streptomycin injection—1 gram daily injection I M for 3 weeks Dihydrostreptomycin in the same dose for the same period has been found equally useful in patients who do not tolerate streptomycin

Para Amino Salicylic Acid (P A S) Given by mouth in 3 gm dose 4 times a day for 3 weeks alone or together with streptomycin is useful 50 p c P A S ointment has been found very helpful in dressing tuberculous ulcers and 20 p c solution in dressing a tuberculous sinus

Isoniazid (I N H) 200 mg daily in divided doses by mouth is helpful particularly in cases which are resistant to calciferol therapy Isoniazid is one of the newer forms of treatment of lupus vulgaris As the tubercle bacilli acquire resistance to the drug, streptomycin is given to obviate that possibility and also to act as a synergist Isonicotinic acid hydrazide 300 mg by injection has been found to be very helpful even in the treatment of the primary tuberculous complex of the skin

In tuberculous skin disease the following method has been found to give very encouraging result Streptomycin 1 g by I M injection daily for 3 weeks together with by mouth P A S 3 g 4 times daily From the fourth week to 3 months Calciferol 50 000 i u by mouth twice daily and then Isoniazide 50 mg thrice daily for 3 months

liver extract is given by intramuscular injection in dose of 2 c c on alternate days and is sometimes helpful, (5) Multivitamin is given by intramuscular injections, (6) Calciferol may be given in dose of 50 000 I U twice daily for a long time keeping a check on blood calcium level, E S R and albumin and cast in urine, (7) Isoniazid in dose of 150 mg orally per day for a period of 3 months can also be used, (8) Cortisone may be given orally starting with 25 mg every 6 hours in the first week and gradually reducing in about 6 weeks time (8) Cortisone may be locally infiltrated in the lesions, (9) Hydrocortizone (Roussell) may be applied locally

development of oval, skin coloured subcutaneous nodules over the trunk which are not symmetrically distributed

Besides the skin lesions there may be present lymphadenities with or without the mottling x ray change in the lungs and rarefaction and cyst-formation of the bones of the hands and feet Enlargement of the spleen and liver may be present together with enlargement of parotid glad, lacrimal gland, mammary gland and testis

Diagnosis (1) Symmetrical arrangement of skin coloured or brownish infiltered plaque or nodules on the face and trunk, (2) Biopsy-histopathology shows collections of epitheloid cells with few giant cells in the dermis, (3) Kveim test is positive is sarcoid, (4) Tuberculin test is negative

Differential diagnosis (1) Lupus vulgaris, (2) Lupus erythematosus, (3) Syphilitic cutis, (4) Leprosy, (5) Leukaemia cutis, (6) Eosinophilic granuloma of face, (7) Lymphocytoma of face

Prognosis Is good but sometimes patients develop tuberculosis and die

Treatment Prophylaxis is to avoid tuberculous infection or getting immunized with BCG vaccine

Curative (1) Arsenic should be given as Liq Arsenical in dose of m 3 in water thrice daily and for a long time may be continued also, (2) Calciferrol in dose 100,000 I U orally every day or 600,000 I U. by intramuscular injection weekly may be tried, (3) Isoniazid has also been advocated in dose of 3 mg. per Kilogram body wight for a period of 6 months, (4) Crude

pin head in size and is papular (3) Blood picture shows slight leukopenia and increase in the number of mononuclear cell (4) Histopathology shows subcorneal vesicle formation and swelling of vascular endothelium in dermis

Differential diagnosis (1) Chicken pox (2) Small pox (3) Urticaria (4) Drug rash (5) Pityriasis rosea (6) Id reaction to fungus or bacteria

Prognosis Good in uncomplicated cases

Treatment Prophylaxis—Injection of (1) Convalescent serum (2) Pooled serum of several adults (3) Anti measles serum (Bengal Immunity, Calcutta) (4) Gumma globulin in dose of O 1 c c per pound of body wieght may be given by intramuscular injection

Curative—Alkaline mixture with sedative To prevent complication Aureomycin or Terramycin may be used orally for 4 to 5 days or Penicillin crystalline G injections Locally liniment Calamine with 1 p c Hydrarg Ammon may be used Gumma globulin is given by intramuscular injection in dose four times the prophylactic dose

SMALL POX

This is also called *variola*

Definition Is an acute infectious and highly contagious skin disease characterized by bullous rash all over the body with grave systemic reaction

Etiology Is due to a virus Age—affects all ages but adults are commonly affected Sex—both sexes

Signs and symptoms Incubation period is 12 days Prodromal [illegible] backache with high

CHAPTER IX

VIRUS DISEASES OF THE SKIN

Common viral skin diseases in the tropics are

1 Measles,	6 Herpes zoster,
2 Small pox,	7 Molluscum contagiosum,
3 Chicken pox,	8 Condylomata accuminata
4 Herpes simplex,	9 Warts,
5 Herpes progenitalis,	10 Tropical bubo

Measles.

Definition is an erythematous pin point to pin head sized lesion all over the body with systemic reaction and coryza affecting the infants and children

Etiology Virus is responsible Age Common in children and infants Sex—both the sexes suffer

Signs and symptoms Incubation period is 11 days There is always a prodromal symptom characterized by circumoral flush Later on pin point to pin head sized skin lesions appear all over the body with fever conjunctivitis rhinitis, bronchitis and inflammation of the buccal mucous membrane called Koplik s spot The rash is papular and is red in colour

Clinical types are —

(a) Mild type,
(b) Toxic type,
(c) Haemorrhagic type
(d) Congenital type
(e) Pre natal type which is said to be responsible for the development of birth marks in the newborn

Diagnosis (1) Infants or children having coryza bronchitis, conjunctivitis rhinitis and Koplik s spot within the mouth, (2) Rash is red and pin point to

Curative—Nothing is known Aureomycin or terramacin capsule 250 mg every 6 hours for 8 days together with vitamin C (200 mg) by mouth 4 times a day may be given Alkali mixture with a sedative is very valuable In the pustular stage and later on also lotio Condy's bath twice daily followed by application of 2 p c Ung Hydrag Ammon is helpful For complications Penicilline crystalline 'G' in 5 lacs dose is injected every day for 5 to 7 days Lesions, before they are completly healed, should be touched with undiluted Condy's lotion 4 times daily

CHICKEN POX

This is also called *varicella*

Definition Is an acute infectious skin disease characterized by the formation of vesicular rash and a mild systemic reaction.

Etiology Is due to a virus The virus is said to be the same as the virus of herpes zoster

Age all ages but common in children Sex both sexes

Signs and symptoms Incubation period is 14 days There is no prodromal symptom During the first three days vesicular rash appears on the head face and the trunk and then spreads down the extremities Rashes come out in successive crops The vesicles rupture and crust forms in 10 days Itching is a prominent symptom Scratching gives rise to secondary infection Constitutional symptom is either absent or very mild

D... ... icular rash developing on head ... ld constitutional symptoms, (2)

fever. By the fourth day the temperature falls and the symptoms disappear but reddish papular rash comes out. The papules soon become vesicular and after four days become pustular. Site of the rash is the face and the upper part of the body and the upper extremities. Lesions may appear on the mucous membrane and conjunctivae with a red halo round each vesicle. The second rise of temperature with toxicity again appears. The pustule has a thick wall. The lesion may be discrete or may be confluent in nature The pustular lesion soon becomes umbilicated. The lesions scale off from the eleventh day leaving pitted scars called "pock mark." Pain all over the body and restlessness are the prominent symptoms. Sometimes the vesicle may be filled with blood when it is called *haemarrhagic small pox* and is a grave condition.

Diagnosis: (1) Prodromal symptoms, (2) Second rise of temperature when the rash becomes pustular with toxic symptoms, (3) Histophathology shows multinucleated vesicle with ballooning degernation of cells in the stratum mucosum

Differential diagnosis: (1) Drug Rash (2) Chicken pox.

Chicken pox	Small pox
1. *Prodromal symptoms*—Nil	1. Preset
2. Appearance—with rise of temp	2 after temperature falls
3 Lesions—vesicles are superficial	3 Vesicles are deep seated
4. Distribution of rash—head, face and body	4 Face and exposed parts

Prognosis: Is grave in non-vaccinated cases than in vaccinated cases.

Treatment: Prophylaxis—pox vaccination gives a long lasting immunity. Repetition of vaccination is necessary during an epidemic.

symptoms, (4) May be associated with pneumonia or malaria and (5) Histopathology—In the epidermis vesicle is formed in the stratum mucosum due to coagulation necrosis In the dermis there is oedema with dilatation of blood vessels and perivascular infiltration.

Differential diagnosis

(1) From Herpes Zoster

Herpes Simplex	Herpes Zoster
1 Does not follow nerve routes,	1 Develops along nerve routes,
2 Recurrent,	2 Non recurrent,
3 Painless,	3 Painful,
4 Leaves no scar	4 Leaves scar,
5 No post herpetic neuralgia,	5 Post herpetic neuralgia often results,

(2) From Chancroidal ulcer of genitalia by the history of exposure Ito Reinsterna s test, and by the presence of B ducrey on smear examination

(3) From Syphilitic ulcer by the history of exposure, presence of T pallidum by the dark field examination of smear and Blood Kahn and W R tests

Prognosis good

Treatment Touch several times during the day and night with spirit rectificatus and dress with dusting powder where possible May be sealed with collodion When vesicles have ruptured 1 p c Ung Hydrag Ammon locally is helpful Repeated vaccination with small pox vaccine is of great value in recurrent herpes simplex Aureomycin may be used as ointment and orally as capsule 250 mg every 6 hours for 5 days

Vesicle is mutiloculated and is found in the stratum mucosum Intranuclear inclusion bodies called "Lipschutz bodies" and ballooning degenertion of cells of stratum mucosum is found

Prognosis *Good*

Treatment Prophylaxis-avoid contact with a patient of varicella or herpes zoster

Curative-Aureomycin or terramycin capsule (250 mg) by mouth every 6 hours for 8 days together with *Elixir* Vitamin B Complex may help Locally lotio Condy's bath followed by 2 p c Ung Hydrag Ammon application

HERPES SIMPLEX

Difinition Is an *acute* or *chronic* skin disease characterized by vesicle formation anywhere on the skin or mucous membrane

Etiology Is caused by a virus Is found associated with cold, pneumonia and malaria Virus may remain latent for many years and may get reactivated by trauma

Signs and symptoms Grouped vesicles which *are pin-head or larger in size are found on slightly* red skin Common sites are face and the genitals but may be found anywhere on the body Occur also on the mucous membrane of mouth May rupture forming crusts and getting well without *leaving scar in a week s time* May recur after sometimes In patients with atopic eczema herpes simplex virus causes an extensive vesicular disease with high fever and lymphadenitis when it is called *Kaposi's varicellifrom eruption*

Diagnosis (1) Grouped vesicles on slightly red skin, (2) Recurrent nature of the lesion, (3) No *constitutional*

HERPES PROGENITALIS

Definition Is a skin disease characterised by the development of recurrent grouped vesicles on the body of the glans penis and also on the vulva

Etiology Is due to a virus

Age—adults are affected Sex males commonly

Signs and symptoms Several tiny vesicles with erythematous rings appear in crops on the penis and over the glans penis in males and upon the labia majora and minora, vesitbule and perinium of females It is painful and causes burning sensation When vesicles rupture undermined ulcers appear

Diagnosis Recurrent grouped vesicles on the genitalia of adults

Differential diagnosis (1) Chancroid, (2) Multiple hard chancre, (3) Fixed drug rash

Prognosis Good

Treatment Dusting powder is helpful consisting of calomel and zinc oxide in equal parts 3 p c Aureomycin ointment is also advocated

HERPES ZOSTER

Difinition This is an acute skin disease characterised by pain fever and vesicle formation on one side of the body

Etiology Infection of the posterior nerve root ganglion by a virus and is supposed to be the neurotropic strain of small pox virus May occur at any age

KAPOSI'S VARICELLIFORM ERUPTION

Definition : Is an acute contagious, bullous disease with fever of infants due to a virus.

Etiology : It is due to the herpes simplex virus

Age—children are affected specially but adults are rarely affected. People with eczema suffer usually.

Signs and symptoms : The disease occurs suddenly. Grouped bullous lesions appear on the face with fever. The lesions may then spread on the scalp and down the neck and appear in sucussive crops. Lymphadenitis also occurs. Sometimes lesions are umbilicated. Lesion undergoes involution, crust forms and exfoliates Diarrhoea may occur. Anuria also develops. Otitis media and corneal ulcer also develop.

Diagnosis : (1) Sudden appearance of grouped vesicles on face with faver in a patient suffering from some skin disease, (2) examination of blood shows leucopenia, (3) histopathology shows intraepidermal vesicle formation which later becomes subepidermal. Nuclear inclusions are present. Infiltration in the dermis is dense.

Prognosis : Is good.

Treatment : Prophylaxis is that a patient of skin disease *should not be vaccinated nor should* come in contact with a patient of herpes simplex

Curative is to give sulphadiazine tablet one every 6 hours for 5 days with Mist. Alkali, Penicilline crystalline 'G' is also injected I.M. in O. 2 Mega unit twice daily for 5 days. Vitamin C (200 mg) given orally 4 times a day. Locally *Liniment* Calamine with 1 p.c. phenol is advocated and 2 p.c. Ung. Hydrag. Ammon at night.

rarely get pain Small vesicles appear (Fig No 113) for a week on red patches of the skin along the course of a nerve and crusts form May be on the trunk or on the limbs There may be successive crops (Fig No 114). The course is about 2½ weeks In the herpes zoster of face the first branch of the sensory division of the trigeminal nerve is usually affected and may result also in corneal ulceration

Scars are either hyperpigmented or depigmented It is not recurrent The bulla fluid is serous to start with becoming purulent later on but sometimes may be haemorrhagic also Neuralgic pain usually persists for a long time Idiopathic herpes occurs *without* any cause

Types of herpes zoster are (i) Herpes frontales, (ii) Herpes ophthalmicus, (iii) Herpes facialis, (iv) Herpes pectoralis (v) Harpes abdominis, (vi) Idiopathic herpes

Diagnosis (1) Unilateral painful distribution of hyperaemia or vesicle (2) Grouped vesicle on red skin, (3) Fever and malaise (4) Non recurrent, (5) *Course* about 2½ weeks, (6) Heals leaving pigmentation or depigmentation at the site of the lesions (7) *Histo-*pathology *shows in the* epidermis a vesicle formation due to ballooning degeneration and intranuclear inclusion bodies are *formed* Bulla is formed *in* the stratum mucosum In the dermis there is *oedema* with dilatation of vessels and cellular infiltration Virus also causes inflammation and even *necrosis of the* posterior spinal root ganglion of the sensory nerve

Differential diagnosis (1) Herpes simplex, (2) Varicella

both sexes. May be seen associated with arsenic therapy and leukaemia cutis.



Fig. No. 113
Herpes Zoster
(Case of Captain S. N. Roy)



Fig No. 114
Herpes Zoster
(On the sacral region and thigh of a child aged 6 years)

Signs and symptoms : unilateral intercostal redness with hyperaesthesia *may be the earliest manifestation* or there may be fever with pain on the part. Children

rarely get pain Small vesicles appear (Fig No 113) for a week on red patches of the skin along the course of a nerve and crusts form May be on the trunk or on the limbs There may be successive crops (Fig No 114) The course is about 2½ weeks In the herpes zoster of face the first branch of the sensory division of the trigeminal verve is usually affected and may result also in corneal ulceration

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Differential diagnosis (1) Herpes simplex, (2)

Prognosis Usually good but sometimes may destory the eye or lesions may became gangreneous when the prognosis is grave.

Treatment (1) Pain is relieved by Aspirin or by Potassium Bromide with Sodium Salicylate, (2) Pituitory extract 0 5 to 1 0 c c. I. M. injection once daily for 2 to 3 successive days before the development of vesicles aborts or (3) Dihydroergotamin (Sandoz) is injected I M in 2 c c dose twice daily for 2 to 3 days, (4) *Antibiotic may be used—Aureomycin* 250 mg. capsule by mouth with water 6 hourly for 4 days have proved of great value During aureomycin therapy, vitamin-B complex should be administered also 4 times daily and thereafter also for a week *thrice daily after food,* (5) *Vitamin* B^{12} *in large* doses (500 microgram daily) is helpful to allay the post-herpetic neuralgia when injected intramuscularly for two weeks, (6) Cortisone Roussell may be given in severe *cases and particularly in old patients The dose should be* 25 mg by mouth every 6 hours for 3 days with vitamin-B complex and vitamin C for the first week, 12 5 mg. with vitamin B complex and vitamin C every 6 hours for the next *3 days,* and then 5 mg *6 hourly for* 3 *days, 5 mg 8 hourly for a day* and then 12 hourly on *the last day (7) Locally Lotio Calamine* with 1 p c Phenol application every hour is advocated, (8) X-ray application to the posterior nerve root ganglion of the *spinal cord is helpful Ultra-Violet* exposure *is sometimes* helps

MOLLUSCUM CONTAGIOSUM

Etiology It is a virus disease and is mildly contagious Commonly seen in children Incubation eriod

is several months Brick shaped virus particle of molluscum contagiosum has been demonstrated by electronic microscope



Fig No 115
Molluscum co tag osum
(On the un b l cus)

Signs and symptoms Pin head to pea sized nodular growth on the healthy skin The nodule has an umbilication at its top It has a pearly appearnce Grows very slowly Common sites are face hands chest back adomen (Fig No 115) and may be on genitals (Fig No 116) Fresh lesions develop on a scratch mar (Koebner s phenomenon po itive) Lesions are neither painful nor itchy

Diagnosis (1) Pearly tumour with umbilication (2) Commonly in the pediatric age (3) Histopathology multiple bodies grow down from the epidermis There

is degeneration of the cells of the stratum corneum forming a homogeneous eosinophilic inclusion body called "Molluscum body". Dyskeratosis is a feature.



Fig. No 116.
Molluscum Contagiosum on the genitalia of an infant
(Case of Major B. Chakrabutty)

Differential Diagnosis : (1) Infective wart and (2) Epithelioma.

Prognosis : Good.

Treatment : Locally each tumour should be picked off with a toothed desecting forceps and the base is cauterized with phenol. Electro-cautery can also be used. 50 p c. Trichloracetic acid application after puncturing is *helpful. Antibiotic (Aureomycin 250 mg. orally 6 hourly* for 5 days with Vit-B Complex orally) has definite value in molluscum contagiosum. Sulphapyridine in dose of O' 5 gm. orally every 4 hours for 5 days is also advocated.

CONDYLOMA ACCUMINATA.

Definition : It is a skin disease characterised by the formation of a collection of thin and elongated warty growths due to a virus.

Etiology: It is due to a virus and is predisposed by an irritating discharge.

Age—found in adults. Sex—seen in both sexes.

Signs and symptoms: It is associated with gonorrhoeal discharge in males and vaginal discharge in females. Pregnancy is also sometimes associated in in famales. Warty growths like cauliflower is found around the vulva, around the corona glandis and sometimes in the crural regions (Fig. No. 117). The colour of the warts are greyish yellow.



Fig No 117
Condyloma accuminata

Diagnosis (1) Cauliflower-like growth around the vulva, corona glandis, crural region in adults, (2) Histopathology shows dilatation of vessels in the dermis with acanthosis of the stratum mucosum of epidermis.

Differential diagnosis (1) Condyloma lata, (2) Cancer.

Prognosis · Good.

Treatment: Prophylaxis is to keep parts clean and daily bath. Curative is to paint the lesion with 20 p. c.

podophyllum resin in liquid paraffin protecting the skin and washing it. Surgical removal with a diatherapy needle may be done.

WART

It is also known a *Verruca* or *Infective wart.*

Etiology : Is caused by a virus.

Signs and symptoms : Classification : (i) Verruca vulgaris, (ii) Verruca digitata, (iii) Verruca filiformis, (iv) Verruca plantaris, (v) Verruca plana, (vi) Condylomata acuminatum.

Warts are skin coloured, circumscribed papillary growths. *May occur on the skin all over the body* (Fig. No. 118). There is no pain or tenderness



Fig No 118
Infective warts
(Case of Dr K C Kandhari)

except in verruca plantaris where pain is felt on walking. Infective wart is commonly seen on the face of children.

Diagnosis (1) Skin coloured papillary tumour (2) Histopathology shows hyperkeratosis acanthosis and elongation of the rete ridges

Differential diagnosis (1) Molluscum contagiosum

Treatment Removal of the warts by electro cautery and Carbon dioxide freezing Sometimes repeated daily applications of 20 p c Acid Salicylic in Collodion can cure the condition

TROPICAL BUBO

This is also known as *lymphopathia venereum* or *lymhogranuloma venereum*

Definition Tropical bubo is a venereal disease caused by a virus and is characterized by a genital sore followed by the enlargement of inguinal glands

Etiology This is due to a virus infection acquired by sexual contact but rarely even by extra genital contact Incubation period is 3 weeks May affect both sexes Age—adults are the victims

Signs and symptoms After sexual contact grouped vesicles appear on the mucous membrane of the genitalia They are painless and discharge colourless serous fluid During this time there may be fever with anorexia Within a week the lesions heal without scar formation 2 weeks after the appearance of the genital lesions the inguinal lymph glands of one side only get enlarged and tender Later on the lymph glands become fixed (Fig No 119) to the skin and get slowly enlarged The skin over the gland becomes red and sinuses

are formed through which seropurulent fluid comes out The condition may heal after several months leaving elephantiasis of the genitalia or the patient may develop virus meningitis or meningo-encephalitis, polyarthritis and rectal strictures may also develop.



Fig. No. 119
Tropical Bubo
Lymphogranuloma venereum
(Case of Dr. B. N. Banerji)

Diagnosis : (1) History of sexual contact followed by herpetic lesion on the genitals accompanied with fever and malaise and later on unilateral inguinal lymphadinitis, sinuses and elephantiasis of the genitalia, (2) Frei's test positive, (3) Biopsy—histopathology shows dense infiltration with plasma cells in the dermis together with epitheloid cells and giant cells.

Differential diagnosis : Granuloma venreum.

Prognosis : Is fair these days with modern treatment.

Treatment : Sulphapyridin for 10 days, Aureomycin for 3 to 6 weeks with Vitamin-B complex, Streptomycin for 6 weaks, Anthiomeline 20 injections Superficial X-ray therapy may be tried. Surgical excision is often helpful.

CHAPTER X

PARASITIC DISEASES OF THE SKIN

The common parasitic diseases found in the tropical dermatological practice are mainly

(A) Vegetable parasites viz fungus (B) Animal parasites viz (i Scabies and (ii) Pediculosis

The common vegetable parasitic infections in general practice in the tropics are those due to Tinea and Actinomycosis

Tinea affects the skin, nail and hair There are three common types of fungi such as (1) Microsporon (2) Epidermophyton and (3) Trichophyton

The disease is called ringworm infection, or fungus infection or Tinea infection

Tinea infection can be classified as follows

(1) Tinea capitis, (2) Tinea barbae (3) Tinea corporis, (4) Tinea cruris, (5) Tinea pedis and Tinea manuum, (6) Tinea versicolour (7) Erythrasma, (8) Favus and (9) Trichonocardiosis axillaris

TINEA CAPITIS

Tinea capitis is also known as ringworm of the scalp Seen commonly amongst prepubertal children but is rare in India Cases are seen in India only in the hills of Darjeeling, Simla and the like When rarely occurs it is seen in the epidemic form in the schools

are formed through which seropurulent fluid comes out The condition may heal after several months leaving elephantiasis of the genitalia or the patient may develop virus meningitis or meningo-encephalitis, polyarthritis and rectal strictures may also develop.



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as it destroys all the hair follicles Itching is one of the symptoms



Fig No 191
Ringworm of Scalp
(Kerion type in a boy aged 6 years)

Diagnosis (1) Wood s light examination—Wood's light is the filtered ultraviolet radiation When the head is examined in a dark room with Wood s light the infected hairs show fluorescence Green with Microsporon audonini and Microsporn canis but bluish with Trichophyton (2) Microscopical examination of infected hair under microscope for fungus, (3) Culture of the infected hair for fungus

Differential dignosis (1) Alopecia areata, (2) Trichotillomania (3) Syphilitic alopecia (4) Seborrhoic capitis and (5) Psoriasis of the scalp

Prognosis Is good except in acute type of Microsporon canis infection because it develops Kerion celsi which results in permanent baldness

Treatment Prophylactic—the patient should not be allowed to go to the school and should protect his head with a cap which should be boiled daily to prevent the spread of infection

Locally (1) Shaving the head should be done for y (2) Washing the head with soap

Common organism is the *Microsporum andonini* which is very rare in India particularly in the planes. *Microsporum canis* is sometimes found both in adults and children only in those who are found closely associated with dogs and cats. Sometimes *Trichophyton violaceum* is also found responsible which produces black-dot lesion.

Signs and symptoms: Starts as a scaly patch on the scalp (Fig. No. 120) where the Microsporum canis infection is present and the condition becomes acute.

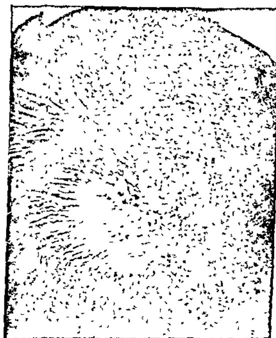


Fig. No. 120

Tinea Capitis

(On the scalp of a girl aged 4 years)

A boggy swelling develops on the head (Fig. No. 121) with multiple openings called *Kerion celsi*. When it gets well permanent baldness is left due to *Kerion celsi*

Differential diagnosis (1) Contact dermatitis (2) Bacterial folliculitis (3) Drug rash (Iodide or Bromide)

Treatment *In the acute stage* 1 p c Lotio Ichthyol application is helpful During the subacute and chronic stages 4 p c Ung Hydrarg Ammon application or Ung Whitfield locally are helpful

TINEA CORPORIS

Tinea corporis produces ringed lesions of erythemato squamous type or only squamous type of lesions are found on the body (Fig No 123 & 124) Causative fungus are Trichophyton and Microsporon Attacks all ages and both sexes Itching is annoying Itching results



Fig No 123
Tinea Corporis



Fig No 124
Tinea Corporis et Cruris

and water every day, (3) Application of an ointment (Containing Hydrarg. Ammon.—gr. XX in Vaseline—oz. 1) after bath and before going to bed at night.

After shaving and washing the head with a bland soap and water application twice daily may be applied (1) Ung. Whitfield (Acid Benzoic gr. 25, Acid Salicylic gr. 15, Vaselin alba oz 1), (2) Sodium propionate or (3) Undecylenic acid.

TINEA BARBÆ

Tinea barbæ is known as the ringworm of the beard. Tinea barbae is the chronic ringworm infection of the hairy part of the beard region (Fig No. 122). Both microspora and trichophyta are causative organisms.



Fig No 122
Tinea barbae
(Kerion type)

Tinea barbae may be (1) Ringed type, like a coin in shape where there are broken hairs with a patch of eczema or (2) inflammatory type which may be pustular or kerion celsi type or may be (3) sycotic type with crusting and broken hairs

Diagnosis : (1) Microscopical examination of an infected hair, (2) Culture from the lesion, (3) Wood's light examination is also very helpful.

the fungus infection of the groin, perineum and upper part of the thighs. The causative fungus is the Epidermophyton inguinale. The lesion produced is commonly a bilateral flexural infective eczema The margin is clear cut with erythematosquamous lesion and is somewhat raised from the surface of the skin (Fig. No 124) The itching is very severe. There may be associated 'Id'reaction all over the body Commonly seen in adults of both sexes.

Diagnosis (1) Skin scraping examination shows the presence of fungus (Fig. No. 125), (2) Culture of the skin scraping for fungus

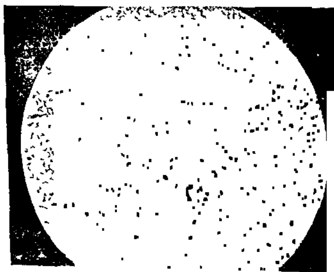


Fig No 125
Microscopic view of fungus
from the skin scraping
(Case of Dr. G. Panja)

Differential diagnosis : (1) Moniliasis, (2) Folliculitis.

in secondary infection with strepto and staphylococci Sometimes multiple gyrate type of lesions appear with granulomatus margin Typical ringed lesion is sometimes found on the body (Fig No 123)

Diagnosis (1) Skin scraping is to be examined on a glass slide with 2 to 3 drops of 10 p c Sodium Hydroxide and covering with a cover slip The slide is warmed and examined under microscope, (2) Culture of the skin scraping for fungus

Differential diagnosis (1) Eczema (2) Seborrhoic darmatitis, (3) Syphilitic cutis (4 Leprosy, (5) Psoriasis

Prognosis is good

Treatment Hygiene is very important The patient should use a bland soap like Margo Soap (Calcutta Chemical) and all the clothings and towels used by the patient should be sterilized daily by boiling for at least one week during the course of treatment Application locally of Ung Whitfield regularly or Undecylenic acid are helpful When there is a localized patch present painting the part with Tr Iodine twice daily or 2 to 5 p c Ung Chrysophanic acid 2 to 5 p c Ung Derobin or 2 to 5 p c Ung Cignolin are very helpful A check should be kept on the urine for albuminuria Ween local application of these medicines produces sensitization the local application should be stopped and Lotio Calamine is to be applied locally for 7 days before treatment is resumed

TINEA CRURIS

This is also known as *Dhobi s itch* Tinea cruris is

From this place it extends downwards and all the interdigital spaces are involved and gives the typical look of 'Haja' There is sodden skin present in the interphalangeal spaces Similarly in the hand one of the interdigital spaces are involved first and all others are ultimately affected This is the common type found in maidservants and in those who keep their hands and feet wet, (2) Another type of lesion is the vesiculobullous type and involves the plams and soles without affecting the interdigital spaces, (3) A type of infection where the skin of the soles become thickened (Fig. No. 127), (4) Sometimes nails of fingers and toes are affected



Fig No. 127
Tinea plantaris
(Case of Dr. G. Panja)

(Fig. No. 128) which become black, thickened and furrowed.

Sometimes the whole palm is involved and becomes chronic with desquamating lesions. Rarely erythematosquamous lesions persist on the palm or sole.

Treatment: Prophylaxis is the scrupulous hygiene. In treating a patient the hygiene must be followed rigidly. Patient's underwear and towels should be boiled every day after bath. In the oozing stage treat as any eczema that is if the oozing is profuse 1 p.c. lotio silver nitrate soaks every hour for 24 to 48 hours Next 1 p.c. aqnous lotio gentian violet painting followed by Ung. Whitfield application for 2 weeks. In intractable cases 1 to 2 p c. Derobin or Cignolin in Acetone may be painted. Undecylenic ointment may be used with advantage.

TINEA PEDIS

Tinea pedis and Tinea manuum is commonly known as Athlete's foot or *Epidermaphytosis.* This is the ringworm infection of feet and hands. 'Haja' or 'Panilagna' are common terms in Bengal and Bihar for this condition.

Trichophyton rubrum is the common fungus but Trichophyton interdigitale is also found.



Fig. No. 126
Tinea Pedis
(Fungus infection in between toes and Lichen simplex on ankle)

(1) Commonest type the commonest site is the fourth interdigital space of one or both feet (Fig. No. 126).

From this place it extends downwards and all the interdigital spaces are involved and gives the typical look of 'Haja' There is sodden skin present in the interphalangeal spaces Similarly in the hand one of the interdigital spaces are involved first and all others are ultimately affected This is the common type found in maidservants and in those who keep their hands and feet wet, (2) Another type of lesion is the vesiculobullous type and involves the plams and soles without affecting the interdigital spaces, (3) A type of infection where the skin of the soles become thickened (Fig No 127), (4) Sometimes nails of fingers and toes are affected



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(Case of Dr G Panja)

(Fig No 128) which become black, thickened and furrowed

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Fig. No. 126
Tinea Pedis
(Fungus infection in between toes and Lichen simplex on ankle)

(1) Commonest type-the commonest-site is the fourth interdigital space of one or both feet (Fig. No. 126).

The hygiene of the feet and hands should be carefully followed Daily washing with soap and water followed by the application of medicines and the patient should boil the shocks gloves and towels daily Patient should better use a separate bathroom

'ID' REACTION

Id Reaction is an allergic reaction to the infection by fungus or bacteria Together with ringworm infection there may be lichenoid rash all over the body and is known as trichophitid (Fig No 1 9) reaction



Fig No 129
Trichophytid
(Id reaction to fungus)

Trichophitid may be sudden and may appear even during the course of treatment Itching may be present Fungal diseases have a tendency *to hypersensitivity* which is an allergic state and purduces superficial inflammation all over the body

Diagnosis (1) Skin scraping for fungus (2) Culture of skin scraping for fungus



Fig No 128
Tinea Unguam

Differential diagnosis (1) Dermatitis venenata or contact *dermatitis* 2) Drug rash (3) Papulosquamous syphilis of palm and sole (4) Acrodermatitis *perstans* tropicalis

Prognosis Good

Treatment In the acute stage it is to be treated with 1 p c aquous lotio Silver Nitrate soaks for 2 to 3 days When the oozing subsides treat with painting of 1 p c aquous gentian violet followed by Ung Whitfield application and the parts to be kept covered by bandaging or by wearing shoes and gloves To be repeated twice daily for a period of two weeks In the chronic stage one of the following may be used (1) Ung Whitfield, (2) 2 p c Ung Derobin or Cignolin (3) Ung Zinc Undecylenate or (4) Castellani's Fuschin paint

Diagnosis (1) Skin scraping when seen with 10 p c Sodium Hydroxide under a microscope Mala furfur is seen in the scraping (2) Culture of skin scraping

Differential diagnosis (1) Depigmented stage of dermal leishmaniasis (2) Seborrhoea, (3) Ringworm and (4) Leprosy

Treatment Improvement of the general health is essential Hygiene is important in the form of boiling of the underwears and towels daily Locally applications of one of the following —

Ung Whitfield 5 c c Ung Sulphuris, local application of 25 p c Sodium hyposulphite aquous solution followed immediately after with 10 p c aquous Tartaric acid solution Sometimes application of Tn Iodi mitis followed by 1 p c aquous silver nitrate is helpful

ERYTHRASMA

Erythrasma is a noninflammatory fungus infection of the type known as *Nocardia* Common sites are axillæ and crural regions Starts as a macular, reddish, circular spot which may be as large as a coin Gradually the colour changes to yellow

Diagnosis Skin scraping shows Nocardia

Differential diagnosis (1) Tinea versicolor and (2) Tinea cruris

Treatment Shaving the hairs and keeping the part clean with soap and water washing are essential followed by daily application of Tn Iodi mitis application or Ung Undecylenic acid or 10 p c alcoholic solution of sodium propionate Hygiene shold be follwed

The 'Id' reaction is caused by the absorption of the fungal toxin in the blood from the site of the lesion which circulates in the blood and comes out in the form of a rash only when the skin is sensitized.

Diagnosis: (1) Associated with the 'id' reaction there should be present ringworm lesion on the body, (2) Skin scraping from the ringworm lesion for fungus is positive, (3) Culture of the scraping from the ringworm lesion is positive and (4) Fungus is absent in 'Id' lesion.

Differential diagnosis: (1) Early secondary syphilis, (2) Drug rash, (3) Pityariasis rosea and (4) Seborrhoea.

Treatment: (1) Locally lotio Calamine soaking for 2 to 6 days followed by 1 p.c. Ung. Acid salicylic application, (2) Antihistaminic drugs are helpful when given in the form of one tablet or capsule (Antistin, Benadryl, Calciluvin or Saudostin) three times daily for 5 days, (3) Vitamin C (200 mg.) tablet 6 hourly for a week is also helpful.

PITYRIASIS VERSICOLOR

Definition: Tinea versicolor is caused by a fungus.

Etiology: Is due to a fungus called *Malassazia furfur*. In the tropics Tinea versicolour and Tinea niagra are due to the same organism

Signs and symptoms: Is seen in all ages and in both sexes. Light brown coloured macular spots which coalesce to form nummular patches or very big sheets. Commonly found on the V of the neck like a Japanese fan. May also be seen on face, axilla, neck, chest, back and thighs. Scales can be seen with a hand lens. Itching is present which may be quite severe at times.

Diagnosis (1) Scutula and mousy odour with alopecia of the scalp (2) Wood's light is helpful

Differential diagnosis (1) Tinea capitis (2) Seborrhoic eczema (3) Lupus Erythematosus

Prognosis Alopecia when produced is permanent

Treatment (1) Hygiene of the scalp, (2) Locally Ung Whitfield or Ung undecelynic acid applications

ACTINOMYCOSIS

Actinomycosis is not a rare disease in the tropics A type which commonly affects the sole is prevalent in South India and is known as *Actinomycosis madurae*

Signs and symptoms The infection starts as a small sore and then an indolent growth develops which is riddled with sinuses Serous discharge with yellow granular bodies exude The granules when pressed with a cover slip on a slide and are seen under microscope shows Ray fungus In culture actinomycosis grows

Differential diagnosis (1) Syphilis, (2) Cancer

Prognosis Should be guarded as it may involve muscles bones and even internal organ and can cause death In an early case the prognosis is fair

Treatment Prophylaxis is to put on foot wear Curative Penicillin cyrstalline 'G 0 5 Mega unit is injected I M daily for 3 to 6 weeks Streptomycin in gram dose may be injected daily for 4 to 6 weeks with PAS orally Aureomycin (250 mg) capsules 6 hourly with Vit B complex 3 to 6 weeks Iodide should be given by mouth for a long time in high dose

FAVUS

Definition : Is a fungus disease caused by Trichiphyton Schoenleini which affects the scalp primarily and also the body.

Etiology : Both sexes are equally affected

Signs and symptoms : Commonly affects the scalp but rarely other parts of the body and the nail. Starts as yellow vesicles. Crust forms which is cup shaped called the *scutulum* leaving a depressed scar. Hair is irregularly affected and grows along the length of the hair. It is a very chronic disease It produces permanent alopecia. Patients emit a mousy-odour. Both the hair and the skin of the scalp (Fig No. 130) are involved.



Fig No 130
Favus of Scalp

Head looks moth-eaten. The body may show erythemato-follicular lesion. Affects both sexes below pube ...

disease produced by the female *Acarus scabies* and is characterized by vesicles

Etiology Scabies is quite common in the tropics It forms about 15 p c of all skin diseases Age it affects all ages but is commonly seen in children Intimate contact with a patient causes infection Common in winter

Sex—Both sexes are equally affected

The Acarus scabii which is also known as Sarcoptes scabii is a minute living mite which is about 1/60th of an inch in size and in shape it is ovoid. Is has four pairs of legs The female causes the disease called scabies It lays its eggs in the skin for which it burrows in the epidermis and as it goes deeper the eggs are left behind The eggs hatch and larvae come out in about 3 days and move on the skin surface The larvae moult into nymph twice and then moult into the male and female in 17 days time After copulation the male dies and the female digs her own grave and she goes deeper in the skin About 50 eggs are laid. The female dies after about 6 weeks The burrows are in length about 1/8th to 1/4th inch Common in winter and during war, femine and the like

Signs and symptoms The lesions are vesicular and sometimes erythemato papular The burrows can be made out with a hand lens on the front of the wrist It is very itchy particularly when in bed The distribution is typical the vesicles may by found along the *anterior axillary folds*, the areola, the umbilicus the linea alba, the inguinal ligaments, the genitalia and the perineum Round the shoulders, round the elbows, front of the wrists and webs of the fingers (Fig No 131) Round

TRICHONOCARDIOSIS AXILLARIS

Definition : Is a kind of fungus infection of the hair of axilla making it lusterless.

Etiology : Is due to the infection of the hair with Nocardia tenuis. Varieties are (a) Black, (b) Yellow, (c) Red. Red and black type usually occur with chromogenic cocci

Signs and Symptoms : Only the cortex of the hair is infected and the hair becomes lusterless in the axilla.

Diagnosis : (1) Lusterless hair of axilla, (2) Examine the hair with 10 p.c. Sodium hydroxide.

Defferential diagnosis : Ringworm.

Prognosis : Good.

Treatment : Shave the hair and apply Ung. Whitefield and hygiene is to be followed.

DERMATOZOONOSES

Zoonosis means the dwelling of living insect in the skin such as scabies, microfilaria, ankylostoma, dracontiasis etc. Epizoonosis means the dwelling of the living insect on the skin and on the appendages of the skin such as pediculosis where the louse lives on the skin and on the hairs.

Common zoonoses are : (1) Scabies, (2) Filariasis cutis, (3) Dracontiasis cutis, (4) Creeping eruption, (5) Myiasis, (6) Ground itch, (7) Amaebic cutis, (8) Oxyuriasis dermatitis and (9) Dermal leishmaniasis.

SCABIES

Definition : Scabies is a contagious a

Treatment Prophylaxis consists of daily bath and washing of clothings regularly which is done as a routine in the tropics hence the incidence of scabies in normaltime is neglible Curative consists of (1) Hygiene—this consists of sterilizing of all linens used by the patients by boiling and the woolen garments by ironing (2) Medicinal—the patients after bath with good scrubbing with soap applies the following ointment for a week and changes his clothings daily

Sulphur ppt	gr 25
Hydrarg Ammon	gr 10
Vaseline Alba	oz 1

Ung for external use all over except face

If the patient follows the hygiene well and rubs this ointment every day after bath he gets well in 5 to 7 days Some patients may return after a week with itching and with erythematous le ions all over the body This may be either reinfection or usually sulphur dermatitis If the distribution is not typical of scabies the patient is treated with liniment Calamine for 2 to 5 days

If a quicker result is aimed at, instead of the above ointment the patient after soap water bath and scrubbing is painted thoroughly from neck to the toe with 25 p c Benzyl benzoit emulsion which is allowed to dry up before putting on clothing In moderate infection one painting is sufficient but in severe infection the patient is painted again next day without bath On the third day the patient takes a bath and puts on clean clothing When the patient complains of pruritus after a week it is generally due to Benzyl benzoit dermatitis or reinfection

FILARIASIS CUTIS

Definition Filariasis cutis is caused by microfilaria



Fig. No 131
Scabies on hands

the gluteal regions, medial side of things, round the knees, round the ankles and webs of the toes. In non-ambulatory patients and young children also on the palms and soles and in infants may be on the face and scalp. Mild constitutional symptoms may develop in scabies infection. Albuminuria may also develop.

Diagnosis : (1) Typical lesions which are erythemato-vesicular, (2) Distribution is typical, (3) Demonstration of a burrow and finding out of a living acarus, (4) Biopsy histopathology shows a bulla on the superficial layer of stratum corneum and a tunnel in the stratum corneum going obliquely down to the stratum granulosum with eggs and the mite and another bulla at the bottom of the tunnel. The two intra-epidermal bullae with the tunnel give the apearance like a dumb-bell. In the dermis there is perivascular infiltration and slight edema

Differential diagnosis (1) Impetigo, (2) Urticaria, (3) Dermatitis herpetiformis, (4) Drug rash, (5) Bullous syphiloderma, (6) Erythema multiforme.

Prognosis : Good,

Differential diagnosis (1) Lichen Planus, (2) Cellulitis

Prognosis Good In ochocerciasis it is grave only when the microfilaria migrates into the orbit

Treatment Prophylaxis consists of using mosquito nets at night and freeing the neighbourhood of mosquitoes and flies Curative consists of using Hetrazen tablet or Benocide tablet by mouth in the following dose one tablet thrice daily for 3 to 6 weeks Surgical removal of the onchocerca nodule and excision of loa loa are done

DRACONTIASIS CUTIS

Definition Is the skin disease caused by the invsion of the hypodermis with the guinea worm

Etiology It is quite common in the tropics The female guinea worm is responsible for the disease The guinea warm embryo is poured form an infected person's foot and is ejected into the water when the patient gets down near a tank The embryo tries to find out a host which is a cyclops In the body of the cyclops it changes its shape and develops a tripartite tail This cyclops when gets into the stomach of a man with drinking water gets digested leaving the guinea worm larvæ in the stomach to develop and migrate to such parts of the body and comes out when in contact with water After the embryos are set free they pass through the stomach wall and copulate and the male dies The female guinea worm takes about a year and half to develop to its proper size of about 25 inches in length and in thickness is like that of a thin earthworm The female guinea worm gradually migrates to the skin with her head forward And can be seen and felt under the skin of legs or back

of different species resulting in dermatitis, lymphedema, nodular swellings, transitory and permanent swellings.

Etiology : Filariasis cutis is caused by *Wuchereria bancrofti* and *malayi* and by *onchocerca volvulus* and by *loa loa*. The microfilaria is transmitted by mosquitoes. Onchocerceiasis is due to Onchocerca volvulus which is transmitted by black fly (Simulieum) whereas loa loa is transmitted by mangrove fly.

Signs and symptoms : The incubation period is very long and is several months. Lymphangitis with erysepelatous skin lesions are the earliest signs. Periodically these conditions recur. Sometimes lymphedema alone is present. Pain in the axillae, groins, and scrotum may be associated with fever. Later on elephantiasis of the extremities, scrotum, vulva and breast may develop. There may be orchitis and hydrocele also. On the elephantoid skin there may appear oozing, cracks, fissures and ulcers.

In onchocerciasis subcutaneous nodules are also found on the scalp instead of elephantiasis. In loasis transient tumours dovelop, of the size of a bettle-nut, anywhere on the body and are known as *Calaber swelling*.

Diagnosis : (1) Examination of blood for Microfilaria and loa loa, (2) Skin scraping for onchocerciasis, (3) Blood picture shows eosinophilia and leucoytosis, (4) Biopsy—histopathology shows epitheloid cells and presence of giant cells may be found in the dermis. Destruction of elastic fibers in the dermis with disappearance of the skin appendages may be found. The microfilaria may be found in the dermis also.

CREEPING ERUPTION

Definition Is a skin disease caused by the migration of the larva of the ankylostoma the gnanthostoma or by the larva of tropical warble fly (Hypoderma)

Etiology Commonly the larvae of Ankylostoma braziliense is responsibe Larvae of gnanthostoma are also responssible They all get attached to the skin and burrow deep After reaching the hypodermis they migrate leaving a serpigenous ulcer Both sexes and all ages are affected in the tropics

Signs and symptoms Erythematous nodules are commonly found on the back and buttocks Gradually the nodules disappear and superficial ulcer with an erythematous border is left which gradually spreads circumferentially and is called the *Creeping eruption*

Diagnosis (1) Typical serpigenous lesion, (2) Site back or buttocks (3) Blood picture eosinophilia, (4) Biopsy histopathology shows atrophy of the epidermis above the parasite and the body of the larva

Differential diagnosis (1) Scabies, (2) Myiasis

Prognosis Good

Treatment Prophylactic treatment is to clean the neighbourhood of jungles and logged areas and regular D D T spraying by the Public Health authorities Putting on proper clothing to avoid exposing the bare body

Curative consists of (1) Painting the part twice daily with a skin antiseptic, (2) applying twice daily on the lesion a paint containing

Acid Salicylic	gr	15
Collodion flexile	oz	1

Signs and symptoms : Found in adults of both sexes. A bulla appears near the ankle which bursts after a day or two. The ulcer is at the opening of the uterus. Sometimes the uterus is prolapsed through the ulcer. From the site of the ulcer a zigzag serpigenous papular elevation may be felt upwards which is the coiled female guinea worm. There may be associated lymhangitis and urticaria.

Diagnosis : (1) Typical site of the bulla or ulcer and the guinea worm can be palpated from the site of lesion upwards to a great length, (2) lymphagitis, (3) when the leg is put in a bowl of cold water the ejection of whitish fluid from the ulcer can be observed, (4) Blood picture will show eosinophilia and leucoytosis, (5) Microscopic examination of a drop of ejected fluid will show guinea worm larvæ, (6) Biopsy—histopathology of the skin will show the body and uterus of the guinea worm.

Differential diagnosis : (1) Chronic sinus due to osteomyelitis, (2) Creeping eruption.

Prognosis : Good.

Treatment : Prophylaxis consits of (a) boiling the *drinking water, (b) Stop infected into get down in* the tank. Curative (a) Before the bulla has formed injection of aquous mercuric chloride solution (1 in 1000) is done at several places on the body of the guinea worm to kill it which gradually gets absorbed in the body, (b) Excision and taking out of the worm, (c) *By dipping* the foot with the ulcer in a bowl when the uterus is *prolapsed* and a stick is passed through it and slowly the worm is pulled out.

of forceps after dropping chloroform or ether on the sore and then the wound is dressed

GROUND ITCH

Definition Is an itching skin disease confined only to the feet and legs and is caused by the larva of ankylostoma in the tropics

Signs and symtoms It is due to the larva of ankylostoma duodenale The larva in penetrating the skin produces minute scratches which are very pruritic in nature Sometimes peticheal haemorrhagic lesions also develop Vesicular lesions are also seen Thus a local erythemato vesicular rash is produced which is very itchy and is called *ground itch* The inguinal glands often get enlarged due to secondary infection

Treatment Consists of prophylaxis use of foot wear Locally lotio calamine with or without Lotio Phenol (1 in 100) may be used Liniment calamine with 1 p c Liq picis carb detergens may be applied An ointment can be applied consisting of

Calamine ppt	dr 1
Hydrarg Ammon	gr 10
Acid salicylic	gr 10
Liq picis carb det	m 10
Vaselin Alba	oz 1

Ung for external use and keep the part bandaged Internally carbon tetrachloride and oil chenopodium are given in a mixture *form* followed by a saline purgative Cristoid (S & Z) is helpful

AMAEBIC CUTIS

Amoebic cutis is the most important and fairly common type of protozoal skin disease found in the tropics

(3) Antimony by parenteral injection is helpful such as Urea Stibemine (Brahmachari) intravenously starting with the smallest dose weekly and giving a total of 2 0 gram or Stibinol '100' (Stibanate) injecting intramuscularly biweekly starting with 0 5 cc and increasing by 0 2 c c. in every injection until 2 0 c c. per injection is given which is repeated 10 times

MYIASIS

Definition Myiasis is a skin disease produced by the larva of a fly

Etiology -The fly may deposit its ova in the wound on the skin where the larvae is hatched out producing myiasis Besides this, larvae of different flies attacks the human skin and complete their full development

Signs symptoms Tender nodule of the size of a pea may develop which changes its place underneath the skin This nodule may become red, suppurate discharge pus and ultimately the fully matured fly

Diagnosis (1) Typical shifting nodular growths on the exposed parts of the body or the presence of maggots in a wound (2) Blood picture shows eosino philia, (3) Biopsy—histopathology shows either a larva or a developed fly covered over by atrophied epidermis

Prognosis Good

Treatment Prophylaxis is to keep the patient with multiple skin lesions under a mosquito net to avoid laying of eggs in sores by the flies or to use well covered garments so that the larvae of flies may not attack the skin Curative maggots are removed with a pair

PEDICULOSIS

Definition Is a skin disease caused by lice and is characterised by itching and impetigenous lesions

Etiology Is found in the tropics Due to better hygienic habits it is a less common disease in the tropics than in temperate climates Scarcely a case is found in the skin out patient of a big teaching hospital in India for demonstration to the medical students

The louse is a blood sucking parasite on man Common varieties of louse infecting the human host are —(1) Pediculosis capitis affecting the scalp, (2) Pediculosis corporis affecting the body, (3) Pediculosis pubis affecting the pubic region

The colour of the louse changes according to the colour of the hairs to camouflage itself The colour may be from pale brown to blackish brown In shape also there is some difference The head louse is longer than the louse affecting the pubic region In size the louse varies from 1 to 4 mm in length The female louse is larger in size than the male louse It has three pairs of legs armed with hooks The eggs are deposited at the roots of hairs and are fixed with a color round the hair root There may be many eggs attached to one single hair Eggs are greyish white in colour and oval in shape Each louse produces about 300 eggs but about 50 p c do not hatch Affects all ages and both sexes

Signs and symptoms The louse may be found on the scalp on the eye lashes and all over the body and over the pubic region Itching is the only symptom Itching produces scratching when coccal infection takes place

Etiology Entamoeba histolytica is the cause of the disease The cutaneous amaebiasis follows the (a) intestinal infection, (b) perineal abscess, (c) drainage of a liver abscess, (c) following the drainage of a ruptured appendix

The cutaneous amaebiasis is always secondary to intestinal amaebiasis

Signs and symptoms Chronic granulomatous ulcer develop round the anus, buttocks, perineum or around drainage of a liver absecess There may be slight discharge on the granulation tissue History of amaebiasis

Diagnosis (1) Examination of stool for amaebiasis, (2) Typical sites of the granulomatous ulcers, (3) Histopathology will show the presence of granulomatous change in the dermis and the presence of a vegetative form of Entamaeba histolytica in the dermis

Differential diagnosis (1) Cutaneous gumma, (2) Dermal leishmaniasis, (3) Lupus vulgaris.

Prognosis Good

Treatment Prophylaxis is to treat with emetine any case of liver abscess before doing drainage

Curative is to inject daily emetine hydrochlor gr 1 intramuscularly for 9 days Together with the injections the patient is given by mouth Aureomycin (250 mg) capsule daily, one every 6 hours with Vitamin B-complex, for 9 days The cutaneous lesions are dressed with 1 p c aquous solution of emetine hydrochlor or 3 p c. Aureomycin ointment

Dermal Leishmaniasis is in next chapter

causes pruritus ani and vulvae as well as lichenified papular itchy dermatitis round the anus or around the anus and vulva in children

Etiology Signs and symptoms The female thread worm deposits her eggs on the anal muco cutaneous junction When the egg is deposited and when the larvae are hatched out it produces intense itching of the anus Due to the scratching the skin round the anus and perineum become excoriated and thickened as well as brownish red in colour During scratching ova gets into the nails and the child when puts its fingers in the mouth reinfection takes place Insomnia develops

Diagnosis (1) Pruritus in the anal region of a child (2) Excoriation and lichenified brownish red lesion around the anus or around the anovulval regions in a child (3) scraping from the anal region when examined under the microscope boat shaped enterobious eggs can be seen

Differential diagnosis (1) Condylomata lata (2) Contact dermatitis (3) Piles

Prognosis Good

Treatment (1) Gentian violet in cachet in dose of 2 5 mgm each one such thrice daily for a child for a week is helpful (2) Cristoid (Sharp and Dhome) child dose followed by a saline purgative May be repeated after a month (3) Nails of the child should be regularly clipped off and 1 p c Hydrarg Oxidi flavum ointment is applied to the fingers at night The child should be in bed pyjama at night

and impetigenised lesions develop. There may be petichеal haemorrhages also. Lymphadenitis may be found. Insomnia may develop.

Diagnosis : (1) Itching, (2) Demonstration of the louse or the nit (egg of louse), (3) Impetigo, (4) Microscopic examination of the hair with a nit or the louse.

Differential diagnosis : (1) Impetigo, (2) Eczema.

Prognosis : Is good. Is grave when there is an incidence of a case of typhus or relapsing fever or trench fever near about.

Treatment : Prophylaxis consists of cleanliness and regular bath. In an endemic region or when there is a case of pediculosis in the family it is advisible to (1) Dusting of all clothing with 1 p. c. DDT powder or 5 p. c. pyrethrum extract.

Curative is done by using ordinary powder such as Pearl powder (Bengal Chemical) with 5 p. c. DDT· for dusting of beds and clothings every day and the patient should take bath with a bland soap followed by application of 4 p. c. Ung Hydrarg Ammon for the whole body but for the scalp and eye lashes 1 p. c. Ung Hydrarg Oxidi flavum is advocated. Sometimes Lethane hair oil for the scalp is useful. 1 p. c. Gammexane in alcoholic solution can also be used after shampooing of the scalp. One application may be sufficient. Lorexane concentration (Imperial Chem Pharm) is such a preparation. Aerosol bomb is advocated for body louse infection. 25 p. c. Benzyl benzoit emulsion painting is used for the infection of body and public regions.

OXYURIASIS DERMATITIS

Definition : Oxyuris vermicularis (thread worm)

Varieties commonly seen are · (1) Hypopigmented lesion, (2) Erythematous lesion, (3) Nodular lesion and (4) Xanthmatous lesion.

Signs and symptoms Hypopigmented lesion of dermal leishmaniasis may occur on any part of the body but are usually seen on the face, around neck, back, chest, (Fig No 132) arms, forearms, hands and thighs. Pin point macules appear which become larger in size and coalesce to form big sheets. Hypopigmented patches (Fig No. 133) are macular are non-itchy and non-scaly. Few months



Fig No 132
Dermal Leishmaniasis
(Hypopigmented Stage)

after the appearance of hypopigmented patches butterfly-erythema appears on the face. The wings of the butterfly are represented by the erythematous lesions on malor regions and the body of the butterfly is on the nose. This stage may persist for some months Butterfly erythema becomes prominent after the patient has

CHAPTER—XI

GRANULOMAS OF THE SKIN.

Common granulomas in the tropics are :

(1) Dermal Leishmaniasis, (2) Syphilis, (3) Yaws, (4) Pinta, (5) Tuberculosis, (6) Leprosy, (7) Granuloma anulare. (8) Foreign body granuloma. and (9) Granuloma inguinale.

DERMAL LEISHMANIASIS.

Two types are common in the tropics and particularly in India. The type which is common in Bengal, Bihar, Orissa and Assam is popularly known as '*Brahmachari's Disease*' or Post Kala-azar Dermal Leishmaniasis. The other type which is seen in other parts of India with a dry climate is known as "*Delhi Boil*" or Oriental Sore. Distribution of Kala-azar and oriental sore are not seen together. These diseases are transmitted by sand-fly called *Phlebotomus argentipes*. The causative organism is a Leishmania.

Dermal Leishmaniasis : This disease is a sequelae to Kala-azar and about 2 p. c. of Kala-azar patients develop post kala-azar dermal leishmaniasis. Commonly found in lower India, Eastern Pakistan, Ceylone, Burma, Shyam and China. The organism responsible is called Leishmania donovoni and the transmitting sandfly is known as Phlebotomus argentipes. Dermal leishmaniasis occurs as a host-parasite adjustment. Dermal leishmaniasis usually occurs one to two years after Kala-azar. It is not related to oriental sore.

in the dermis In the erythematous stage there is oedema in the dermis with dilatation of papillary vessels



Fig No 134
Dermal Leishmaniasis
(Nodular stage)

with perivascular infiltration Parasites can be found in this stage in the dermis In the nodular stage there is thining of the epidermis with flattening of the papillae There is not marked change in the papillary layer of the dermis but the reticular layer of the dermis shows dilatation of vessels with perivascular macrophage proliferation Large number of parasites are found in these macrophages In the xanthomatous stage no L. D body is seen (6) Complement fixation test using Leishmania bodies as an antigen is found positive quite early and can even be used as a check to prognosis and follow up Patient should not be considered cured until this test is negative

Differential diagnosis (1) Lupus Erythematosus (2) Lupus Vulgaris (3) Leprosy, (4) Leucoderma (5) Pellagra and (6) Xanthoma Tuberosum

Treatment Antimony is the only treatment

exposed himself in the sun for sometimes. This erythematous stage is followed by nodular stage on face first (Fig No. 134) and then on other parts of the body.



Fig. No. 133
Dermal Leishmaniasis
(Hypopigmated Spotage)

Nodules are deep brownish in colour and are soft to the feel. The stage persists for many years and may or may not finally develop into the Xanthomatous stage Xanthomatous stage is very rare. In the xanthomatous stage the patient develops yellowish plaques which resemble xanthoma tuberosum.

Diagnosis : (1) History of long continued fever or having had treatment for Kala-azar about a year or two back, (2) Aldehyde test is negative, (3) Antimony test is negative, (4) Skin snip smear is positive for L-D bodies when stained with Leishman stain, (5) Biopsy is stained with Hæmatoxylin and Eosin and the histopathology shows in the hypopigmented stage decrease of melanin in the stratum basalis with dilatation of papillary vessels. Parasite may or may not be seen

Diagnosis (1) Smear from lesion shows leishmania tropica, (2) Culture of smear shows leishmania tropica

Differential diagnosis (1) Gummatous ulcer, (2) Desert sore, (3) Tuberculous ulcer, (4) Diphtheretic ulcer.

Treatment Dressing with an antiseptic is usually done Base of the ulcer may be infiltrated with 1% Atebrin solution or 1% Berberine sulphate Dressings with an ointment containing Zinc oxide dr 1/2, Acid salicylic-gr 10, Hydrarg Ammon gr 10, Vaseline Alba oz 1



Fig No 135
Oriental Sore
(Case of Dr S C Desai)

SYPHILODERMA

Syphiloderma is the commonest manifestation of syphilis and is caused by Treponema pallidum.

In the acquired cases syphiloderma develops about 2 weeks after infection and is called "Hard Chancre" Genital and there may also be extra genital hard chancres on lips, fingers, and anal margin

Urea stibamin (Brahmachari Research Lab. Calcutta) starting with 0.01 gram in 2 c.c. pyrogen free distilled water is injected intravenously after examining the urine for the presence of albumin and also blood for agranulocytosis. *Total dose should be 2.5 gram per course.* Injections may be given twice weekly with high protein diet and citrous fruits. This course is to be repeated 6 to 10 times with intervals of 1 month. Other Antimony preparations may be used both I. M. and I. V. Stibanate (Gluconate) 2 c.c. inj. I. M. biweekly. Total dose 30 c.c. repeating 5 to 6 times with one month's interval.

Toxicity of antimony—vomiting, giddiness, cold clammy skin and even signs of collapse. Pulse becomes rapid with headache, discomfort in chest, joint pains and even jaundice. *Adrenalin hydrochlor (1 in 1,000) should* be injected I. M. immediately with the development of any signs of toxicity and thereafter 500 mg. Vitamin C intramuscularly or with I.V 25% Glucose 25 c. c. Liver extract (crude) I. M. inj. biweekly 12 such.

Oriental Sore : Known as Delhi boil or tropical sore. Found commonly in upper India and Western Pakistan and it is *not a sequelae of viseral* Kala-azar. Oriental Sore is found on the exposed parts of the body and face may be first affected (Fig. No. 135) with induration and a dirty base. *In size varies from ½" to 1" diameter* and is almost circular in shape. Starts as a papule on which scales appear. Crusting takes place soon and the ulcer breaks down and slowly extends peripherally. Ulcers heal with a slightly depigmented depressed scar. The sore is inoculable and usually gives protection against reinfection.

Gummatous syphiloderma occurs anywhere on the body (Fig. 136) but are commonly seen on the face (Fig. No. 137) lower legs, trunk, arms and scalp.



Fig No. 136
Syphilitic cutis
(Multiple gummatus ulcers)



Fig. No. 137
Congenital cutaneous syphilis
(Showing destruction of the bridge of the nose and the upper lip)

Early secondary syphiloderma develops about 2 months after the infection and is characterized by rashes all over the body with glandular enlargement. There is bilateral enlargement of the cervical, axillary, epitrophlear and inguinal glands which present a painless, nodular and indiarubbery feel. Rashes are pleomorphic in type that is a mixture of various kinds as follows: (a) Macular-this may again be (i) Roseolar and (ii) Pigmentary in nature. (b) Papular-this may be subdivided into (i) Squamous, (ii) Erythemato-squamous, (iii) Vesicular, (iv) Hypertrophic, (v) Ulcerative, (vi) Mucous patches such as Condylomata lata.

Important characteristic of rashes are: (1) Distribution is symmetrical and bilateral, (2) Configuration-tendency to get arranged in circles, (3) Colour-primary rashes are pinkish but secondary rashes are brownish red, (4) Induration present in the early secondary rash and is absent in early primary rashes.

Late syphilodermic rashes are as follows:

(1) Pigmentary syphiloderma-macular lesion is common on palms and soles of white and black in colour. May rarely occur over the neck called "collar of Venus." Atrophic changes may occur, (2) Nodular syphiloderma-nodules are circular, central necrosis may appear giving rise to a "punched out" ulcer or the nodules may remain so for years, (3) Squamous syphiloderma is seen commonly on palms and soles. This is true nodular syphilide but produces scaling and is dull red, (4) Gummatous syphiloderma gradually becomes larger in size. Central softening occurs and gets adherent to the skin above. The skin breaks down and forms a "punched out" ulcer with a "wash leather" base.

changes of fingers and toes, (6) Syphilitic wig—is the excessive growth of hair in congenital syphilis Syphilitic wig may be followed by patchy alopecia called "moth eaten" alopecia, (7) Generalised adenitis is commonly seen in congenital syphilitics (8) Dactylitis, osteochondritis, craniotabes, Parrot s nodules and "hot cross bun' appearance of the skull are commonly seen

Diagnosis (1) Dark ground examination of smear from the ulcer for Treponema pallidum, (2) Fontana's stain for T pallidum, (3) Wassermann Reaction, Blood W R is pesudo possitivé in (i) Leprosy (ii) Psoriasis, (iii) Chronic malaria, (iv) Infective mononucleosis (v) Diabetes and (vi) Physiological condition like pregnancy and (vii) Cancer Hence it is advisable to do (4) Kahn Test of blood, (5) Histopathology shows infiltration with plasma cells in the dermis with endothelial swelling

Differential diagnosis (1) Hard chancre should be differentiated from (a) Traumatic ulcer, (b) Chancroidal ulcer, (c) Scabies, (d) Herpes progenitalis, (2) Roseolar syphiloderma from (a) Measles, (b) Drug Rash, (c) Pityriasis rosea, (3) Pigmentary Syphiloderma from vitiligo, (4) Squamous syphiloderma from (a) Ring worm, (b) Tuberculoid leprosy, (5) Erythemato squamous syphiloderma from (a) Psoriasis (b) Seborrhoea, (6) Vesicular syphiloderma from (a) Scabies (b) Impetigo, (c) Pustular acne vulgaris, (7) Hypertrophic syphilo derma from (a) Yaws and (b) Pemphigus vegetans, (8) Ulcerative syphiloderma from Ecthyma, (9) Condylomata lata from condylomata accuminata and papilloma

Treatment Should be preceeded by the (1) Examination of Urine, (2) Examination of Blood W R

Congenital Syphiloderma is characterized by macular, papulo-squamous or bullous lesions on the palms, soles, buttocks and on face. Associated with the skin manifestations there may be (1) "Saddle nose", (2) Laryngitis, (3) Rhagades-deep fissuring in the line of the normal skin folds which on healing leave linear fissures, (4) Circumoral nodulo-c utaneous is commonly seen (Fig. No. 138)



Fig. No. 138
Congenital syphilitic cutis
(Annular syphilide in
a girl aged 8 yeers)

Commonly on the ano genital (Fig. No. 139) mucocutaneous junctions, angles of the mouth, (5) Onychia-nail

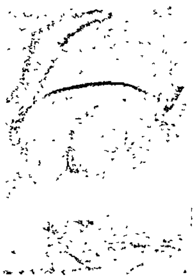


Fig No. 139
Congenital syphilitic cutis
with condylomata lata anum
(Boy aged 3 years)

patient may be declared cured Repeat courses at intervals of one month

Arsenicals commonly used are Acetylarson N A B and Thiosermine (Brahmachari Research Lab Calcutta)

Course consists of weekly intramuscular injection in empty stomach of Thiosermine 0 15 gram one injection 0 30 gram one injection 0 45 gram one injection and 0 60 gram seven injections Total 5 0 gram per course Urine is to be examined for albumin before every injection

Bismuth preparations commonly used are colloidal Bismuth metal in O 2 gram intramuscular weekly injections for 10 weeks making a total of 2 O gram Mercury is used with Iodide in mixture Liquor Hydrarg Perchlor m 15 Potassium Idide gr 5 Tr Cinchonaco m 6 Aqua oz 1 Ft mist for a dose after food thriced aily

Adjuvant It is always beneficial to give the patient high protein diet with plenty of green vegetables and oranges to protect the liver as a prophylaxis against post arsenical intoxications

For Leprosy and Tuberculosis see Chapter VIII

YAWS

It is also called *Frambesia tropica*

Definition It is a chronic non venereal skin disease characterized by various skin lesions caused by Treponema pertenue

Etilogy It is found all over the tropics In India it is only seen in the States of Assam It is caused

(3) Examination of Blood Kahn, (4) Examination of Blood for total counts of R. B. C. and W. B. C. differential count of W. B. C. and Haemoglobin p.c., (5) When possible blood is examined for Vitamin C estimation of fasting blood. It has been observed that when the blood Vitamin C is lower than 0.4 mg. per 100 ml. of blood the patient is likely to develop post-arsenical complications during arsenic therapy.

The treatment should be the combined Penicillin-Arsenic-Bismuth-Mercury therapy.

Penicillin should be given for 10 days by the injection of aquous crystalline penicillin "G" 2.5 lacs twice daily at 12 hours interval with no other treatment or 0.5, Mega unit is injected intramuscularly once every day. In 10 days the total penicillin dosage is 5 Mega unit. (1 Mega unit is 10 lacs unit of penicillin). Antihistaminics and Vitamin C togeher orally or parenterally may be used in penicillin drug rash which may occur as a complication to the antibiotic therapy.

From the 11th day of treatment the following course is given. Arsenicals by injection weekly for 10 weeks, Bismuth by injection weekly for 10 weeks. Mercury and Pot. Iodide by mouth for 14 weeks in form of a mixture.

Followed after a month by the examination of (a) Blood W. R. and (b) Blood Kahn Tests.

If tests are negative repeat 3 courses of Arsenic-Bismuth-Mercury and follow-up the case for 2 years more with blood and clinical examination 4 times a year. When everything is negative at the end of 2 years after completion of the anti-syphilitic therapy the

tartiary stage ulceration of the skin called "gumma and sometimes circinate type of squamous lesions may be seen Depigmented or hyperpigmented patches Juxta articular nodules goudou and mutilation of rose and nasopharynx called *Gangosa* may be seen

Diagnosis (1) Typical lesion on the skin, (2) Treponema pertenue on dark field examination, (3) Blood W R pseudo positive (4) Biopsy histopathology shows acanthosis of stratum mucosum where treponema pertinue may be found Infiltration with round cells and plasma cells in the dermis

Differential diagnosis (1) syphilis (2) Ringworm, (3) Scabies (4) Lichen planus (5) Seborrhoea, (6) Lupus vulgaris (7) Leprosy

Prognosis Good except in very advanced stage

Treatment Prophylaxis is to avoid contact with an infected patient Curative Every ulcer must be covered (1) Acetylarsone (Stovarsol) O 25 gram tablet thrice daily for a week by mouth, (2 Penicillin crystilline O 5 Mega unit in aquous solution is injected intra muscularly every day for 2 weeks Arsenic Bismuth injection Thiosermine (Brahmachari Calcutta) O 15 gram one O 30 gram one O 45 gram one O 60 gram to be injected weekly intramuscularly after urine exami nation and Bismuth O 2 gram is injected intramuscularly once every week Open wounds should be dressed with 4 p c Hydrag Ammon ointment

PINTA

Definition Is a chronic non venereal skin disease caused by *Trepona carateum*

by **Treponema pertenue**. *It affects all ages and both* sexes Existing ulcer in the skin is necessary for infection. Incubation period is 3 to 6 weeks.

Sings and symptoms: It has three stages called (1) Primary stage, (2) Secondary stage and (3) Tartiary stage. It starts in the primary stage as a single macular *lesion on the skin called "Yaws spot". A single* cauliflower-like granulomatous lesion on the skin is called "*Mother Yaw.*" The secondary stage comes 3 months after the primary stage. Maculo-papular or maculo-squamons lesions which appear on the planter surface mainly and nodular growths (Fig. No. 140) on face

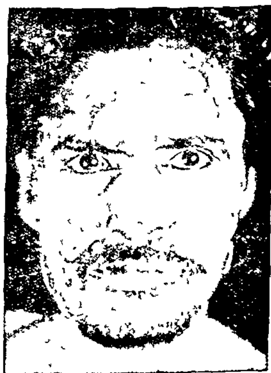


Fig No. 140
Yaws
(Secondary Stage)
(Case of
Dr. P. Damodaram)

and body which is called the "*Crab yaw.*" Sero-purulent discharge is present. Joint pain is called "yaws pain" and itching are common symptoms at this stage. he

Etiology Quite common in the tropics Cause is not known Both sexes and all ages are involved

Types (1) Ordinary type (2) Giant type

Signs and symptoms Papular and skin coloured half pea sized lesions with a tendency to arrange in the form of a ring with a diameter of ¼ ½ to ¾ inches called ordinary type while rarely very large sized lesions are seen called giant type There is no itching and no pain Single lesion seldom occurs except in the giant type but in the ordinary type the lesions are about half a dozen in number Site commonly found on the knuckles of fingers and are also seen over the back of the wrist round the knee round the gluteal regions and the ankles Rarely may be found at other places like scalp

Diagnosis (1) Ringed skin coloured lesion on the back, hand elbow knee ankle joints without any itching or pain (2) Histopathology shows degeneration of the connective tissues and presence of epitheloid cells in the dermis

Differential diagnosis (1) Erythema multiforme, (2) Leprosy, (3) Syphilitic cutis, (4) Lupus vulgaris

Prognosis is fair rarely cured

Treatment Locally may be applied in an ointment containing

Acid Salicylic gr 10, Crude coal tar dr ½ Vaselin alba oz 1

X ray therapy is also advocated High dose of vitamin A is also useful When all other treatment proves useless multiple incisions on the ring down to the dermis is helpful

Etiology: It is found in certain parts of tropics. It is rare in Asia but has been reported from Africa. Affects adults of both sexes. Incubation period is a week.

Signs and symptoms: Two tages such as (1) Primary and (2) Secondary. In the primary stage erythemato-squamous lesions are seen. Lymphadenitis may be present. Secondary stage appears about one year after infection. Erythemato-squamous lesions are mainly found on the face and extremities. On the plam and soles and also on the dorsum of feet and hands papular pigmented lesions alternating with vitilaginous macular lesions are seen which is known as the late **dyschromic stage.**

Diagnosis: *(1)* Vitilaginous macular *lesions alternating* with pigmented macular lesions on the hands and feet in an adult, (2) Blood W. R. positive, (3) Biopsy histopathology shows microabscesses and acanthosis with degeneration of the stratum basalis. In the dermis there may be found *Treponema c rateum* with solid masses of chromrtophores.

Differential Diagnosis: (1) Leucoderma, (2) Syphilis.
Prognosis: Good.

Treatment: Penicillin crystallin 'G' aquous solution intramuscularly is injected O.5 Mega Unit daily for 15 days. Arsenic-bismuth may be used also.

GRANULOMA ANULARE

Also known as ringed eruption or lichen annularis.
Definition: [Is a skin disease of unknown cause and is characterised by skin coloured ringed lesions.



Fig No 141
Granuloma Inguinale
(Case of Dr B S Verma)

Diagnosis (1) Chronic granulomatous unlcer in the genitalia spreading either to the groins or to the perineum or both in adults (2) Skin test of Anderson positive (3) Histopathology shows in the dermis dense small round cells large histiocytic cells with Donovan bodies within them

Differential diagnosis (1) Cutaneous syphilis, (2) Chancroid, (3) Tropical Bubo from Psendo bubo

Prognosis Fair

Treatment (1) Antibiotic such as Penicillin injection 0 2 Mega unit twice daily by intramuscular injection for 2 weeks, (2) Aureomycin capsule (250 mg) every 6 hours for 2 weeks, (3) Streptomycin gram 1 injected intramuscularly daily for two weeks, (4) Achromycin or Iloticin may be useful, (5) Locally 3 p c aureo mycin ointment is advocated

FOREGN BODY GRANULOMA

Definition : Is a granuloma due to the introduction of a foreign body in the dermis.

Etiology : Common forign body is the wax introduced into the *skin of the left hand* accidentaly by shoe-makers in the tropics, thorn or a fish bone. Painless signs and symptons nodular growths on the skin of hands or anywhere on the body. No suppuration or erythema seen.

Diagnosis : (1) Painless, non-suppurating indolent growths nodular (2) *Histopathology shows the* wax by special stain.

Differenital diagonosis : from other granulomas.

Prognosis : Good.

Treatment : Excision.

GRNULOMA INGUANLE

Definition : It is a chronic contagious skin disease characterized by ulcerating granuloma of the genitalia and groin.

Etiology : Sex—occurs in either sex. Age—young adults are commonly affected in the tropics. It is acontagious and is said to be of venereal origin Causative organism is an intracellular body called *Donovan body.* This is a gram-negative bipolar body.

Signs and syptoms : A granulomatous *ulcer (Fig No.* 141) on the external genitalia which gradully spreads towards the groin above and towards the perineum below. It takes the from of subcutaneous nodule called **pseudobobo**. The spread is very slow.

of different shapes and are macular (Fig No 142) Mongolian spot and supernummary ear and super numary finger are also birth marks



Fig No 142
Pigmented Nevus
or birth mark
(Covering infrascapular
region and back of arm)

Sometimes the moles grow hairs on them and are called hairy naevus or Naevus piolosus It is said



Fig No 143
Giant Pigmented Nevus
Pilosus
(Bathing Trunk type)

CHAPTER XII

TUMORS OF THE SKIN.

Tumuors of the skin are mainly of four different types: (1) Benign Epidermal, (2) Benign Dermal, (3) Precancerous and (4) Cancer.

Common benign epidermal tumours are: (1) Naevus, (2) Milium, (3) Sebaceous cyst, (4) Chondrodermatitis Nodularis Chronica Helicis, (5) Adenoma Sebaceum, (6) Multiple Benign Cystic Epithelioma and (7) Cylindroma.

Naevus—This is the common mole. Every human being has moles. There are always multiple moles on the body. Some people have many moles others have just few here and there adding to the beauty when on the face. With the growth of age the moles increase in number. There may or may not be any mole at birth but may appear any time after birth.

Hence naevus may be classified into (a) Birthmark and (b) Mole. Moles are of various types such as: (1) Pigmented mole, (2) Pigmented hairy mole, (3) Verrucous mole, (4) Giant mole, (5) Naevus linearis, (6) Junction naevus.

Moles are about the size of a pin-head or half pea-sized generally. They are either macular or slightly papular. Moles are usually completly black in colour. Moles occur anywhere on the skin of the body. Sometimes a mole is quite large in size and is called a "Birth mark." Birth marks are rectangular oval or

Rarely the mole is unilateral and is confined to one side of the body when it is called **Naevus linearis** (Fig No 146) This is an unilateral mole and may be confined only to one of the extremities or it may run from the ankle and proceed upwards along the medial side of the leg and thigh and reach the ischial tuberosity and then up the gluteal region along the side of the body to the axilla of the same side and down the medial side of the arm and forearm ending at the front or back of the wrist or some times passing over the dorsum of the hand ending infront of one of the knuckles



Fig No 146
Naevus linearis

Junction naevus is the one which undergoes malignancy

Diagnosis (1) Pigmented mole is either macular, papular, verrucose, linear or hairy, (2) Size may be pin head or half pea sized commonly but sometimes it is of a giant type when it covers either the whole of the trunk or parts of limbs or face, (3) Does not undergo malignacy except those which are junction naevi, (4) Histopathology—Epidermis is thin Below the epidermis groups of naevus cells and sometimes giant cells are found Neavus cell is oval with a large nucleus In the verrucous type there is hyperkeratosis with acanthosis and elongation of the rete pegs together with groups of naevus cells

that naevus pilosus never goes malignant Sometimes they are very big and cover the whole of the trunk and are called bathing trunk type of neavus pilosum or giant type (Fig No 143) Not uncommonly verrucoid growths are found on a pigmented mole and is called **Naevus Verrucosum** (Fig No 144 & 145)

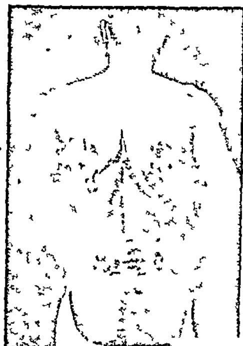


Fig No 144
Naevus Verrucosum

Fig No 145
Pigmented nevus

Differential diagnosis (1) Dermoid cyst and (2) Molluscum contageosum,

Prognosis Good

Treatment Surgical removal

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

It is a nodular painful condition of the helix of the ear Single or multiple nodules of the size of a pea on the helix are found Wrestlers and boxers get it due to the trauma on the ear but rarely occurs without trauma The nodule is attached to the cartilages of the ear When ulcerates it becomes persistent Never goes malignant Commonly seen in men

Diagnosis (1) Situated on the helix of the ear, (2) Painful lesion (3) Biopsy—Acanthosis is present in the epidermis Dermis shows inflammation and infiltration with round cells and some giant cells There is degeneration of collagen and fibrous tissue also The underlying cartilage shows inflammation

Differential diagnosis (1) Epithelioma, (2) Senile keratosis and (3) Eczema

ADENOMA SEBACEUM

Found in both sexes Is often hereditory Disease starts in childhood Pin head to half pen sized nodules appear on the face (Fig No 147 & 148) They are yellowish in colour but some are reddish when fine blood vessels are seen on the nodules Associated abnormalities may also be present such as warts, naevi, pigmented

Differential diagnosis: (1) Warts, (2) Molluscum contagiosum, (3) Basal cell carcinoma, (4) Melanoma.

Prognosis : Is good when a growing naevus is excised.

Treatment : Moles occuring near the places of trauma should be removed by excision. Plastic operation is advised for gaint naevus. When naevus is growing in size it should be removed. Junction naevus should be removed.

MILIUM

Skin coloured pin-head sized lesions situated on the face particularly on the forehead and around the eyes. Often seen in children but may occur at any age. It is neither tender nor painful. It never ulcerates and remains as it is for years.

Histopathology shows a horny cyst with a thin epidermis. The horny mass looks like onion.

Differential diagnosis : (1) Acne comidone, (2) Molluscum contagiosum.

Pronosis : good

Treatment : Incising and scraping the horny cyst.

SEBACEOUS CYST

Sebaceous secretion is poured into the pilo-sebaceous follicle. The cyst is formed by the occlusion of a hair follicle. It is an oval tumour and is soft and elastic. Reacton is present. Cyst is yellowish and semisolid in consistency. Sometimes a cyst becomes infected. May occur anywhere on the body. Multiple sebaceous cysts occur on the scrotal skin which may undergo calcification.

Differential diagnosis (1) Dermoid cyst and (2) Molluscum contageosum,

Proguosis Good

Treatment Surgical removal

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

It is a nodular painful condition of the helix of the ear Single or multiple nodules of the size of a pea on the helix are found Wrestlers and boxers get it due to the trauma on the ear but rarely occurs without trauma The nodule is attached to the cartilages of the ear When ulcerates it becomes persistent Never goes malignant Commonly seen in men

Diagnosis (1) Situated on the helix of the ear, (2) Painful lesion, (3) Biopsy—Acanthosis is present in the epidermis Dermis shows inflammation and infiltration with round cells and some giant cells There is degeneration of collagen and fibrous tissue also The underlying cartilage shows inflammation

Differential diagnosis (1) Epithelioma, (2) Senile keratosis and (3) Eczema

ADENOMA SEBACEUM

Found in both sexes Is often hereditory Disease starts in childhood Pin head to half pen sized nodules appear on the face (Fig No 147 & 148) They are yellowish in colour but some are reddish when fine blood vessels are seen on the nodules Associated abnormalities may also be present such as warts, naevi, pigmented

spots. Sometimes there is a history of mental disease, epilepsy, heart disease or visceral growths May be familial.



Fig. No. 147
Adenoma sebaceum
(Boy aged 7 years with history of epilepsy)

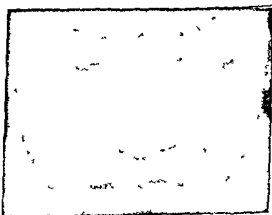


Fig. No. 148
Adenoma Sebaceum
(Girl aged 8 years)

Diagnosis: (1) Early onset, (2) Nodules on face, (3) Histopathology-shows hyperplasia of the hair follicles with hyperplasia of sebaceous gland [illegible] blood vessels

Differential diagnosis (1) Acne vulgaris, (2) Acne rosacea, (3) Colloid milium, (4) Molluscum contagiosum, (5) Multiple benign cystic epithelioma

Prognosis Is never cured

Treatment Removal of lesions is the treatment of choice X ray therapy is also helpful

MULTIPLE BENIGN CYSTIC EPITHELIOMA

Pin head to pea sized yellow nodules occur on face at pubertal age Lesions are symmetrically arranged

Diagnosis (1) Situated on the face, (2) Age of onset is at puberty, (3) Histopathology show proliferation of the cells of stratum basalis

Differential diagnosis Cylindroma

Prognosis Good

Treament Removal of the tumours surgically

CYLINDROMA

Commonly known as turban tumour Occurs o the scalp In size vary from a pea to an agg In number may be single or multiple and the colour is that of the skin Very slowly increases in size and rarely becomes a very big tumour looking like a turban on the head Sometimes there is a history of injury to the head There is often associated alopecia seen Often familial Turban tumours are derived from the coils of sweat gland

Diagnosis (1) Situated on the scalp, (2) Skin coloured tumour of the size of a pea to that of an

agg on head, (3) Histopathology- shows masses of basal cells and hyaline degeneration side by side.

Differential diagnosis : (1) Granuloma pyogenicum, (2) Sebaceous cyst.

Prognosis : If untreated may undergo malignancy.

Treatment : Removal by operation.

COMMON BENIGN DERMAL TUMOURS

They are : (1) Scar, (2) Keloid, (3) Fibroma, (4) Neurofibroma, (5) Neuroma, (6) Lipoma, (7) Glomus tumour, (9) Osteoma, (10) Lymphangioma and (11) Angioma.

SCAR

Is also known as cicatrix. Scar is the new formation of connective tissue in the skin as a result of a skin disease or trauma.

Signs and spmptoms : Scar is at level with the in. It is generally of the colour of the skin. car may assume any shape. It is smooth and shiny. here is no hair on the scar. Occurs at all ages d in both sexes.

Diagnosis: (1) Smooth, shiny, hairless patch of tissue on level with the skin, (2) Histopathology-absence of hair follicle and sweat glands. There are bundles of interlacing connective tissue with no blood vessels.

Complication : Scar may become hypertrophied and results in keloid formation.

Prognosis : Good.

Treatment. No treatment is necessary *until* complication develops.

KELOID

Definition Is a tumour arising from the fibrous tissues of the dermis

Etiology Heredity has some influence Cause is not known Trauma is an exciting factor but sometimes chronic inflammation of the hair follicle is also responsible Some think that the keloid begins by hyperplasia of round cells in the adventitia of arterioles

Classification 2 types (1) Idiopathic and (2) Secondary Age—may occur at any age Sex—both sexes are equally affected

Signs and symptoms Starts as a nodule on the normal skin but usually on a scar (Fig No 149)



Fig No 149
Post acne Keloid

This nodule raises the skin above it and generally enlarges in size. Sometimes becomes slightly red and tiny processes proceed from its base like the crow's claw (Fig. No. 150). It is hard to the feel.



Fig. No. 150
Keloid

Diagnosis : (1) Growth which is hard to the feel and of skin colour, (2) Claw-like processes at the base of the tumour, (3) Histopathology—Hyperplasia of the connective tissues of the dermis. Cellular elements are present in a new keloid but are absent in an old one.

Prognosis : Good with treatment. Some disappears spontaneously

Treatment Repeated injection of Hyaluronidase is helpful in small keloids. But for keloid of very big size excision and radiotherapy. The usual procedure for a moderate sized keloid is to give X-ray therapy in suberythema dose twice at weekly intervals foll ed by thema excision and after the wo

doses of X ray exposure at weekly intervals Sometimes radium application is slso helpful

FIBROMA

Etiology Is not known

Commonly seen in elderly people at climacteric Found in both sexes

Signs and symptoms Single or multiple pedunculated and soft growth of skin colour seen commonly on face neck or other parts of the body In size like that of a pea

Diagnosis (1) Skin coloured single or multiple pedunculated soft growth and in size like a pea (2) Histopathology shows fibrous tissue proliferation

Prognosis Good

Treatment No treatment is necessary Sometimes it is excised from the base of the peduncle and the base is cauterized

NEUROFIBROMA

Known as Von Recklinghausen s disease Is a condition characterized by the development of multiple tumours Skin develops a peculiar pigmented clour

Etiology Not known

Seen in both sexes but is commonly seen in males in the tropics Middle aged males are commonly seen with multiple neurofibromatosis and some are pedunculated Some think it to be due to some change in the germ plasm and it starts from intra uterine life

This nodule raises the skin above it and generally enlarges in size. Sometimes becomes slightly red and tiny processes proceed from its base like the crow's claw (Fig. No. 150). It is hard to the feel.

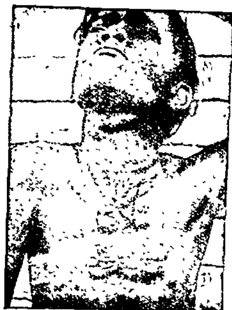


Fig. No. 150
Keloid

Diagnosis : (1) Growth which is hard to the feel and of skin colour, (2) Claw-like processes at the base of the tumour, (3) Histopathology—Hyperplasia of the connective tissues of the dermis. Cellular elements are present in a new keloid but are absent in an old one.

Prognosis : Good with treatment. Some disappears spontaneously.

Treatment : Repeated injection of Hyaluronidase is helpful in small keloids. But for keloid of very big size excision and radiotherapy. The usual procedure for a moderate sized keloid is to give X-ray therapy in suberythema dose twice at weekly intervals followed by excision and after the wound heals two more suberythema

fibrils with the absence of elastic fibres in the tumour With special nerve stain non medullated nerve fibres can be found



Fig No 152
Neurofibroma showing sesile tumors and cafe au lait coloured skin
(Case of Dr K C Sahu)

Complication (1) Mucoid degeneration, (2) Malignant degeneration (sarcomatous change)

Differential diagnosis From (1) Leprosy (2) Mullnscum contagiosum, (3) Multiple cutaneous sarcoma.

Prognosis Bad Sarcomatous changes may occur Mental retardation has also been observed

Treatment Surgical removal when a *single* or a few or big

LIPOMA

Are multiple fatty tumours occuring in the dermis and in the subcutaneous tissue

Signs and symptoms Occurs all over the body from head to foot (Fig No 151)



Fig No 151
Neurofibromatosis

Tumours are soft to the feel In size tumours are pea-sized but gradually become bettle nut in size and even larger weighing about a pound Skin shows (Fig. No 152) one or two depigmented or hyperpigmented patches known as cafe au lait colour

Diagnosis (1) Multiple various sized tumours all over the body in a healthy individual, (2) Histopathology shows loose texture and wavy arrangement of the

Occurs on the extensor surface of the body and are symmetrical in arrangement Sites are face, neck, extremities and trunk, Seen commonly in young adults

Histopathology—shows smooth muscle fibers Muscle fiberes are found to originate from the erectori pilorum mu cle or from the muscular layer of blood vessel

Differential diagnosis From Syringocystoma

Prognosis Good

Treatment Freezing with carbon dioxide snow Excision of those which are pretty big in size

GLOMUS TUMOUR

Is also known as Angioneuro myoma

Signs and symptoms It is a rare tumour Small tumour which is soft to the feel and is painful It may be skin coloured or reddish Found under the nail Pain starts from the finger tip and radiates along the hand and the pain increases with cold There is no metastasis Lentil in size and found in both sexes

Diagnosis (1) Site under the nail of a finger, (2) Painful and pain increases when touched with a cold object, (3) Histopathology shows a network of nerves and arterioles It is an overgrowth of the glomus body There is dilatation of the vessels with hyperplasia of the surrounding cells

Differential diagnosis Neuroma, Melanoblastoma

Prognosis Good after excision.

Treatment Excision of the tumour

Etiology ; Not known. Occurs in both sexes and at all ages.

Signs and symptoms : Soft tumours of varying sizes. May be peasized or the size of a cocoanut. May be single or multiple and is lobulated. Common sites are neck and gluteal region. Found also in the breast of females. Grows slowly. Skin coloured.

Diagnosis : (1) Slow growing skin-coloured lobulated, soft tumour on the neck, gluteal regions, on the breast of a woman, (2) Histopathology shows groups of large fat cells bounded by connective tissue and is encapsulated.

Differential diagnosis : From Dercum's disease which is a painful fatty tumour.

Prognosis : Good as it does not recur after removal.

Treatment : excision

MYOMA

Is a muscle tumour arising commonly from erectoris pilorum.

Etiology : Not known. Found commonly in women. Can occur in any age.

Classification : Two types, (1) Dartoic myoma, (2) Leiomyoma. Dartoic myomas are commoner than the Leiomyoma. They occur on the penis and scrotum of males and on the labia majora and breast of females. Grows slowly. In size starts as a pea becoming an orange and may be pedunculated.

Leiomyoma are hard nodular growths. They are multiple and nonpedunculated. Are pea-sized usually.

serpiginosum, (d) Naevus anaemecus (when blood vessels are absent), (e) Erethyma palmare hereditarium

(2) Cavernous angioma are (Fig No. 153 & 154), (a) Superficial type (Strawberry), (b) Deep type



Fig No 153
Haemangioma Scalp
(Behind left ear)



Fig No 154
Haemangioma
Strawberry ty
(Case of
Dr R N Gupt

OSTEOMA

Is the deposition of bone in the skin and subcutaneous tissue. Osteoma is a new growth. This may be localized or generalized and progressive.

Signs and symptoms : May occur at any age and in both sexes. *As small seed-like papules or in* large plaques can occur on face or anywhere.

Diagnosis : (1) X-ray and (2) Histopathology-typical bony tissue will be found.

Prognosis : Bad as it is progres ive.

Treatment : Excision but is progressive to such an extent that excision becomes impossible.

ANGIOMA.

Is a vascular naevus.

Signs and symtoms : Occurs soon after birth or sometimes several weeks after birth. Occurs as a red tumour which may be on level with the skin or may be elevated from the skin level and is deep. May occur in any organ of the body except in cartilage. May remain stationary or suddenly start increasing in size. Often some disappear by the age of 2 years but others persist throughout life. Occurs in both sexes. It is painless. Cavernous angiomas become larger when the child coughs or cries

Classification :

(1) Capillary angioma are : (a) Spider naevus, (b) Portwine stain (or naevus flammeus), (c) Angioma

(4) Angiokeratoma, (5) Telangiectasia are :
(a) Congenital telangiectasia, (b) Hereditary haemorrhagic telangiectasia, (c) Generalised telangiectasia, (d) Naevus anaemicus.

(6) Naevus Lymphangioma (Fig. No. 157).



Fig No 157
Naevuslymphangioma
(on the left scapular region of a boy aged 10 years)

Diagnosis : (1) Colour of the lesion, (2) Painlessness, (3) Present since birth or some weeks after birth, (4) Histopathology shows dilatation of blood vessels in the dermis and proliferation of newly formed vessels. In the portwine stain type the whole of the dermis is affected. In the cavernous angioma there is dilatation of the capillaries and multiplication of the blood-spaces which have endothelial lining. Lymphagiomata shows cystic swellings in the upper part of the dermis. Angiokeratoma shows great dilatation of blood vessels in the dermis which are filled with blood and with hypertrophy of the epidermis.

(3) Mixed type such as capillary cavernous angioma and Saleıosing haemangioma (Fig. No. 155 & 156)



Fig No 155
Sclerosing angloma
(On the front of right thigh)



Fig No. 156
—Histopathalogy of sclerosing haem:

(4) Angiokeratoma, (5) Telangiectasia are : (a) Congenital telangiectasia, (b) Hereditary haemorrhagic telangiectasia, (c) Generalised telangiectasia, (d) Naevus anaemicus.

(6) Naevus Lymphangioma (Fig. No. 157).



Fig No. 157
Naevuslymphangioma
(on the left scapular region of a boy aged 10 years)

Diagnosis . (1) Colour of the lesion, (2) Painlessness, (3) Present since birth or some weeks after birth, (4) Histopathology shows dilatation of blood vessels in the dermis and proliferation of newly formed vessels. In the portwine stain type the whole of the dermis is affected. In the cavernous angioma there is dilatation of the capillaries and multiplication of the blood-spaces which have endothelial lining. Lymphagiomata shows cystic swellings in the upper part of the dermis. Angiokeratoma shows great dilatation of blood vessels in the dermis which are filled with blood and with hypertrophy of the epidermis.

Prognosis : Good

Treatment : Application of carbon dioxide snow as slush with acetone is helpful in most cases. Repeated twice montly for a fairly long time. Thorium-X application twice monthly for a long time. Application of Radium is helpful in the elevated type Repeated when necessary after 3 to 6 months. Cauterization by chemical or by electro-cautery is helpful in the spider naevus and in telangiectasias. Roentgenray therapy is helpful in the elevated types of angiomas. Excision is helpful in lymhangioma and angiokeratoma.

PRE-MLAIGNANT TUMOURS OF THE SKIN

Malignant cancers of the skin may be divided into two groups such as (1) Precancerous condition of the skin and (2) Cancerous condition of the skin.

(1) Precancerous conditions of the skin : There are certain skin diseases which ultimately become malignant and are called precancerous. 20 p c of precancerous conditions in the tropics become malignant. Pathologically some will show malignant changes but clinically do not present malignant picture. Common precancerous skin lesions are :—(a) *Arsenical* keratosis, (b) Senile keratosis, (c) Cornu cutaneum, (d) Leucoplakia, (e) Bowen's disease, (f) Erythroplasia of Queyrat.

(a) Arsenical keratosis—This is the commonest precancerous skin disease as arsenic is used extensively as a therapeutic agent in medical practice. It results from the ingestion of arsenic orally but rarely also

parenterally The skin change may be observed as early as within 30 days or after 30 years Arsenical pigmentation develops in about 50 p c cases in those undergoing arsenical therapy Arsenical keratosis occurs chiefly on the palm and sole as pin head to small pea like hyperkeratotic lesions When loosely adherent keratotic scales are removed and indurated superficial ulcer is seen This ulcer changes into a malignant lesion either as a fungating type or ulcerating type Sometimes multiple warty growths are seen all over the body Metastasis is rare

Diagnosis (1) History of ingestion of arsenic, (2) Keratotic growth or indolent ulcer on palm and sole or multiple warty growths on the body, (3) Histopathology shows squamous celled epithelioma Dyskeratosis with vacuolization of cells also occur

Differential diagnosis (1) Calosity, (2) Superficial epithelioma of the skin (3) Bowen s disease (4) Minakhi

Prognosis Sometimes lesions disappear without treatment but may persist and undergo malignant change

Treatment Arsenic medication must be stopped Excision of the lesion should be done

(b) Senile keratosis Is characterised by papular pigmented plaques on the dorsum of the hands and face in old people which bleeds when dry crust is removed Lesions are multiple

Diagnosis (1) Old people of over 60 years age (2) Bleeding indolent ulcer or crust covered lesion on the dorsum of the hand or on the face (3) Histoppthology shows hyperkeratosis, acanthosis,

—

thickening of the stratum granulosum. Mitosis may be found in epidermal cells with squamous cell carcinomatous changes prominent.

Differential diagnosis: (1) Arsenical Keratosis, (2) Seborrhoic wart, (3) Xeroderma pigmentosa.

(c) Cornu cutaneum: Is a localized epithelial tumour. Cutaneous horns develop from filiform papilloma, from a wart, from a naevus, from a sebaceous gland and from the mucous membrane.

Cutaneous horn is usually a solitary lesion with an indurated base. The growth is slow but rarely rapid. Common sites are the dorsum of the hands, temples and ears. Rarely it has been found also on the nose (Fig. No. 158), lips, penis and on the trunk. The shape is like a horn-conical, twisted or of any irregular shape. Seen in people over 40



Fig No 158
Cutaneous horn on nose

Diagnosis: (1) Conical, twisted or irregularly hard lesion on the dorsum of the hands or on the face

usually with an indurated base, (2) Age of patient is above 40 years, (3) Histopathology shows hyperkatosis and parakeratosis of the stratum corneum, Squamous-call carcinomatous changes are seen.

Differential diagnosis . Filiform wart or Senile keratosis.

Prognosis : Good when treated properly.

Treatment : Excision is the choice of treatment.

(d) Leucoplakia · Is characterized by whiteness and thickening of the mucous membrane of the tongue, lip or vulva

Sharp marginated white pin head, coin-sized or irregular areas with cracks on the tongue, lip or vulva. Commonly seen in elderly people Leucoplakia of tongue and lips are common in men.

Diagnosis : (1) Advanced age, (2) Typical irregular or circular sharply marginated white plaque on tongue, lips or on vulva, (3) Histopathology shows hyperkeratosis, acanthosis with degeneration of the stratum basalis. Inflammatory cells are present in dermis.

Differential diagnosis . (1) Thrush, (2) Syphilitic leucoplakia, (3) Lichen planus, (4) Lupus Erythematosus.

Prognosis If diagnosed and treated *early* the prognosis is not bad. When malignant changes have taken place the prognosis is grave.

Treatment : Vitamin A therapy in high dose (100,000 i. u.) daily is helpful even when it *has undergone* any malignant change. Vitamin A therapy should be given a

long trial Locally 20 p c solution of Silver Nitrate to be painted When malignancy occurs excision or radio therapy should be tried

(e) Bowen's Disease Characterised by scaly single or multiple plaques on any part of the body. Red, oozing and indolent lesion appears on removing the crust. Can occur at any age and in both sexes

Diagnosis (1) Indolent scaly plaque on the body, (2) Histopathology shows hyperkeratosis, acanthosis with irregular downward proliferation on the epidermis with keratinization and vacuolization of some cells Clumping of nuclei with mitosis are seen Squamous cell epitheliomatous picture is presented

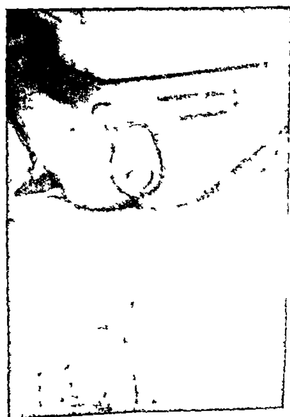


Fig No 159
Erythroplasia of Querrat of penis
(Case of Drs C V Rajam P N Rangiah, P R Azeezullah and J S Serma)

Prognosis . Good when diagnosed and treated early.

Treatment ; Carbon dioxide freezing is helpful in early stage. Excision is the treatment of choice.

(f) Erythroplasia of Queyrat · Slow growing, indolent, red, lesion which occurs on the glans penis (Fig No 159) vulva and on the mucuous membrane of mouth. Occurs in middle aged persons. Regional lymph glands are not enlarged.

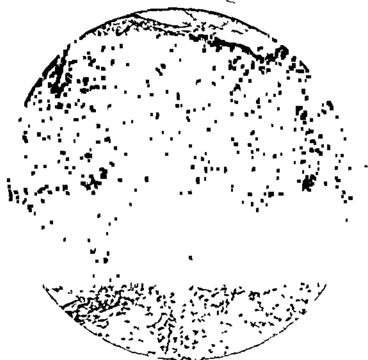


Fig No 160
Histopathology of Erythreplasia of Queyrat
(Case of Drs R V. Rajam, P. N. Rangiah, P. R. Azeezullah and J. S. Serma)

long trial. Locally 20 p. c. solution of Silver Nitrate to be painted. When malignancy occurs excision or radio-therapy should be tried.

(e) Bowen's Disease : Characterised by scaly single or multiple plaques on any part of the body. Red, oozing and indolent lesion appears on removing the crust. Can occur at any age and in both sexes.

Diagnosis : (1) Indolent scaly plaque on the body, (2) Histopathology shows hyperkeratosis, acanthosis with irregular downward proliferation on the epidermis with keratinization and vacuolization of some cells. Clumping of nuclei with mitosis are seen. Sqamuous cell epitheliomatous picture is presented.

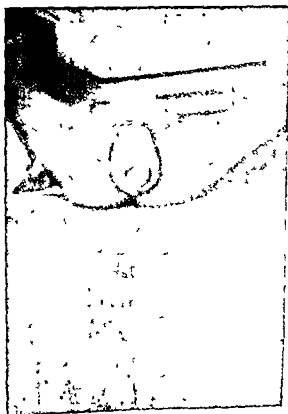


Fig No. 159
Erythroplasia of Queyrat of penis
(Case of Drs C. V Rajam, P. N. Rangiah, P. R. Azeezullah and J S. Serma)

Signs and symptoms: Patients are sensitive to light and particularly to the ultra-violet light. Macular pigmentation on the exposed parts of the body appears



Fig. No. 161
Xeroderma Pigmentosum showing pigmentation and development of *squamous-cell epithelioma.*
(Case of Dr. P. N. Behl)

Diagnosis : (1) Indolent and red genital or buccal lesion in a middle-aged person, (2) No response to any treatment, (3) Histopathology shows dyskeratotic changes with elongation of the rete pegs. There is infiltration in the upper part of the dermis. Squamous cell epitheliomatous changes gradually develops. (Fig. No. 160).

Differential diagnosis : (1) Chronic balanoposthitis, (2) Hard chauere, (3) Soft chancre, (4) Leucoplakia, (5) Leucoplakia of mucous membrane of mouth.

Prognosis : With early diagnosis and treatment it is good. But when advanced the prognosis is grave.

Treatment : In the early stage in case of male genital circumcision and CO_2 application but in the late stage complete exicision is advocated. Complete excision is the treatment of choice also when affecting any other part of the body.

MALIGNANT TUMOURS OF THE SKIN.

Common cancerous conditions of the skin are :

(a) Xeroderma Pigmentosa, (b) Melanoma, (c) Basal-cell carcinoma, (d) Squamous-cell carcinoma, (e) Reticulosis.

(a) Xeroderma Pigmentosa : is a skin disease characterized by pigmentation and development of malignant growths on the skin from the early age.

Etiology : found in the tropics. On an average one case yearly attends a large skin outdoor in India. Cause is not known. It is an inherited skin disease Usually affects only one member in the family. Both sexes are equally affected.

In junction nævus the cell of origin is epidermal Junction nævus is an active nævus hence it is



Fig No 162
Melanoma Sole

potentially malignant The intraepidermal soft nævus may be hyperkeratotic papillomatous and verrucous in look Many contain hairs and hairy moles are benign Some are very large and are called *giant moles*

(2) *Lentigo*—is a very small deeply pigmented mark on the skin Histologically len igo is an early *junction* nævus and may develop into a melanocarcinoma.

(3) Melanocarcinoma—arises *from* a junction nævus or a lentigo rarely from the *normal skin* Starts as a deeply pigmented rapidly growing tumour surrounded by a red hallow Later on the growth ulcerates and small pigmented lesion develop near it and becomes fungating Regional lymph glands are involved due to matastasis Lower extremity is the common site (Fig No 163)

Histologically large nævus cells with *mitotic* fingures and activity at the dermo epidermal *junction* are seen

(4) ie nævus - occurs as an oval, flat blue

and increases each summer. Patchy atrophy of the skin produces perchment skin. Verrucous lesions develop on exposed parts. Photophobia is a prominent feature. In an advanced case the skin presents an exfoliative appearance, keratosis and malignant growths with telangiectasis (Fig. No. 161).

Diagnosis : (1) Dry pigmented skin with photophobia and warty growths on face and hands, (2) Biopsy shows hypertrophy of the stratum corneum with atrophy of the stratum mucosum. Hyperpigmentation of the stratum germinativum. Verrucous lessions show acanthosis and closely packed pigmented cells. Ulcerated lesions show basal cell carcinomatous picture

Differential diagnosis : (1) Senile pruritus, (2) X-ray dermatitis.

Prognosis . Children generally die within a year after diagnosis. Only few cases can reach the adult age. Infants die of marusmus whereas adults die of malignant changes in the skin

Treatment : No treatment is of any use. Sunlight should be avoided. Verrucous lesions and Epitheliomas should be excised. X-ray and radium treatment are not indicated. Vitamin A therapy in high doses (Arovit Roche) should be used for a long time.

(b) Melanoma—Is a pigmented tumour of the skin composed of nævus cells (Fig. No. 162)

Varieties are .—(1) Pigmented nævus, (2) Lentigo, (3) Melanocarcinoma, (4) Blue nævus.

(1) Pigmented nævus are (i) Junction nævus and (ii) Intraepidermal nævus.

Prognosis Is very grave in untreated cases Even in treated cases prognosis is not very good In the slow growing type occurring on face prognosis is not bad Persistent melanuria shows a grave prognosis

Treatment Prophylaxis in junction nævus is removal before puberty Any mole growing near the place of irritation should be removed particularly when a mole develops on the sole and in the subungual regions Benign moles are removed by carbon dioxide and by surgical excision

Curative is the deep and wide excision Regional lymphatics should be removed Since melanomas are radio resistant radiotherapy is useless

(c) Basal cell carcinoma—is also known as Rodent ulcer This is locally malignant and also a growing tumour of the skin

Etiology cause is not known May develop some times after arsenic ingestion

Age—No age is exempt but common after the age of 40 years Sex—Both sexes are equally affected

Signs and symptoms Types are —(i) Button type, (ii) Morphea type, (iii) Cystic type and (iv) Mixed type or Basi squamous type
Growth is very slow

Site—Centre of the face (Fig No 164) but the morphea like and the mixed type are commonly found on the trunk Nodular growth with central umbilication, rolled edge and pearly margin are the characteristic

patch of about half an inch in diameter on the buttock. Histologically occurs in the dermis and do not undergo malignancy.



Fig. No. 163
Melanocarcinoma Sole

Diagnosia : (1) Clinical examlnation, (2) Histopathology and special staining such as Fontana's stain will show melanin and Reticulin stain will show lattice net work in the tumour, (3) Histopathology—the loss of the parallel arrangement of the cells is seen. The nucleus in the cell enlarges in size and mitosis is present. Pigment is found in abundance both in the intercellular spaces and also intracellularly. Elastic tissue is destroyed. Activity in the dermoepidermal nævus and lentigo whereas it is present slightly in junction nævus and is also present enormously in malignant melanoma.

Differential diagnosis: (1) Seborrhoic wart, (2) Sclerosing hæmangioma, (3) Lymphosarcoma, (4) Pigmented papilloma, (5) Blue nævus and (6) Granuloma *pyogenicum*.



Fig No 166
Multiple pigmented basal cell carcinoma
(Case of Major B Chakraborty)

Diagnosis (1) An umbilicated nodular growth or a crusted growth in a person above 40 years of age, (2) Histopathology shows acanthosis in the mucosum. Hyperplasia of the stratum basalis with mitosis of the nucleus in the cells There is no cell nest present (Fig No 167)

Differential diagnosis (1) Syphilis of the skin, (2) Lupus vulgaris (3) Squamous-cell carcinoma and (4) Melanoma

Prognosis Good with treatment

Treatment (1) Excision, (2) Carbon dioxide freezing and (3) X ray therapy.

(d) Squamous cell carcinoma—Is a rapidly growing cancer of the skin which forms cell nests and metasta-

features. Does not usually metastasize. Some basal-cell carcinomas are pigmented (Fig. No 165 & 166).



Fig No 164
Basal cell carcinoma

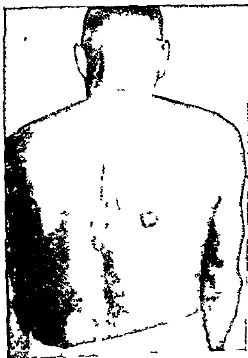


Fig No. 165
Multiple basal cell carcinoma
(Case of Major B Chakraborty)



Fig No 166
Multiple p gmented basal cell carcinoma
(Case of Major B Chakraborty)

Diagnosis (1) An umbilicated nodular growth or a crusted growth in a person above 40 years of age, (2) Histopathology shows acanthosis in the mucosum Hyperplasia of the stratum basalis with mitosis of the nucleus in the cells There is no cell nest present (Fig No 167)

Differential diagnosis (1) Syphilis of the skin (2) Lupus vulgaris (3) Squamous cell carcinoma and (4) Melanoma

Prognosis Good with treatment

Treatment (1) Excision (2) Carbon dioxide freezing and (3) X ray therapy

(d) Squamous cell carcinoma—Is a rapidly growing cancer of the skin which forms cell nests and metastasize early



Fig No. 167
Histopathology of
Basal-cell carcinoma

Etiology: Is unknown. Chronic irritation, X-ray, senile keratosis and cutaneous horn are predisposing factors. Age—middle age. Sex—both sexes

Signs and symptoms: Single warty growth may occur on the face (Fig, No. 168). Edge is everted (Fig. No. 169) and the growth fungates *soon* (*Fig. No* 170 & 171). Regional lymph glands are affected early.

Diagnosis: (1) Single nodular growth, infiltrated at the base, (2) Site face, (3) Regional lymphatic glands are affected, (4) Histopathology-concentric layers

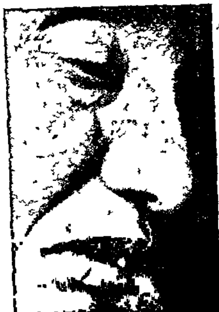


Fig No 168
Squamous cell carcinoma



Fig No 169
Epidermoid carcinoma
lesion on the nape of neck

of keratinized cells called 'cell nest' are always found with hyperplasia of the epidermis and mitosis.

Differential diagnosis (1) Senile keratosis, (2) Lupus vulgaris and (3) Paget's disease

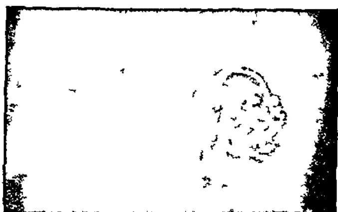


Fig No 170
Squamous cell carcinoma of Ear



Fig No 171
Squamous cell carcinoma

Prognosis In the early stage it is good but after metastasis has occurred the prognosis is grave

Treatment Excision in the early stage Both in the early and late stages application of radium is valuable X ray therapy is of great value.

(e) Reticulosis—is the malignant change found in the reticulo endothelial system Reticulosis cutis includes (1) Leukæmia of the skin, (2) Hodgkin's disease of the skin, (3) Mycosis fungoidis of the skin and (4) Lymphosarcoma of the skin

Signs and symptoms In the early stage itching is most prominent followed by development of rashes which may be urticarial, eczematous and exfoliative This early picture may remain for weeks or months The colour of the skin in the tropics does not change very much except that it darkens In the late stage ulceration occurs

(1) Leukæmia cutis may be subdivided into (i) lymphatic, (ii) myelogenous, (iii) monocytic Leukaemia cutis may be seen at any age Skin lesions are tumours in the skin, subcutaneous haemorrhages even generalised herpes zoster and exfoliative dermatitis of very severe types are found associated

(2) Hodgkin's disease—occurs in both sexes. Pruritus is the main feature Excoriated itchy papules develop all over the body Skin becomes ichthyotic, urticarial rash develops and even exfoliation occurs These symptoms continue for several months to years then the late stage supervenes when nodules appear which ulcerates accompanied with intermittent fever

(3) Lymphosarcoma Is divided into (1) large cell lymphosarcoma, (ii) small cell lymphosarcoma and (iii) Kaposi's multiple idiopathic pigmented sarcoma

Diagnosis: (1) History, (2) Clinical examination, (3) Blood picture, (4) Histopathology shows infiltration in dermis and multiplicity of cells. Dorothy-Reed type of giant eell in Hodgkin's diease, pseudo-giant cell in mycosis fungoides. Mitotic figures are also present. There is multiplication of blood vessels in the dermis in lymphosarcoma.

Differential diagnosis: (1) Eczema, (2) Psoriasis, (3) Urticaria, (4) Exfoliative dermatitis due to arsenic, gold etc. and (5) Pemphigus foliaceus.

Prognosis: Is bad. With modern treatment life may be slightly prolonged.

Treatment: Arsenic by mouth as Fowler's solution in all stages. Nitrogen mustard is helpful in certain cases. Improvement in mycosis fungoides with Para-aminobenzoic acid, in Kaposi's idiopathic sarcoma with ACTH have been observed. Locally antipruritic lotions such as 1 p c. Phenol in Lotio Calamine or 2% Menthol in ointment form are used. X ray therapy is also helpful in some cases.

CHAPTER XIII

COLLAGEN DISORDERS OF THE SKIN

There are some skin diseases in which the collagen ibres of the dermis are mainly affected

Classification (1) Lupus Erythematosus, (2) Sclero lerma and (3) Dermatomyositis

LUPUS ERYTHEMATOSUS

Definition Is a chronic skin condition, sometimes becoming acute which is characterized by adherent scales on erythematous and atrophic base, with dilated mouth of the hair follicle pigmentation itching and is distributed primarily on the face scalp, back, chest and extremities F S C B E

Etiology Is fairly common in the tropics and form about 1 per cent of cases

Common causes are (1) Septic foci (2) Tubreculosis (3) Toxin of strepto and staphylococci (4) Ovarian dysfunction as there is exaggeration of the skin lesion after each pregnancy and bafore menstruation in women (5) Allergic manifestation, (6) Due to exposure to the ultra violet rays of the sun

Varieties (1) Acute and (2) Chronic types Chronic type is subdivided into (3) Chronic discoid and (4) Chronic disseminate

Chronic discoid type of lupus erythematosus is quit common in the tropics and may form about 0 8 pe cent of all skin cases Subacute and acute types are ver rare in the tropics

Age—Commonly starts at the young adult age. Sex-generally seen in women but in the tropics both sexes are equally affected.

Signs and symptoms : The chronic discoid type of lesion may start as a circular pigmented spot anywhere on the face. The spot increases peripherally and becomes pink in colour with a pigmented periphery. There are greyish adherent scales with keratotic plugs on the under surface which fits in the dilated mouth of the hairfollicles. The disease generally starts on both the malor regions (Fig No 172)



Fig. No. 172
Lupus erythematosus

and extends peripherally to get connected over the nose forming a butterfly-like lesion. The wings of the butterfly lying on each malor region with the body on the bridge of the nose. The lesion looks depressed from the surrounding skin due to atrophy.

Site being the face, nose, back, chest and extremities. Atrophy and depigmentation results after cure of the skin lesions. Sometimes nodular growths may be found on the lower lip and chin. The nodular growths are called *Lupus erythematosus hypertrophicus.* Itching is the only symptom.

During the year there may be exacerbation and quiescence of the condition. Rarely ulceration occurs when and patient complains of burning sensation.

The lesion may occur on the tongue as a white patch with a red halo. The lip may be affected where the lesions are like white patches with red halo or there may be crusts with telangiectasia and swelling of lip (Fig. No 173). May occur on scalp causing alopecia.



Fig No. 173
Lupus erythematosus
(lesion on both lips)

Lupus erythematosus lesion may be found on the conjunctiva where it is reddish and edematous in nature becoming atrophic and depressed later on. Burning of the eyes may be the only symptom or there may be itching also.

The chronic discoid lupus erythematosus becomes chronic *disseminated. The lesions on the face look* inflammed and red macular lesions *develope on the body. There may be remission followed by relapse of the*

skin condition within a month or two. Sometimes the lesions may be urticarial, erythema multiforme or macular syphilide in types. Rarely purpuric lesions may be seen. Constitutional symptoms may be present

Systemic reactions - In the chronic discoid type there is no fever or malaise In the chronic disseminate type there may be malaise, fever, arthralgia. In the acute disseminate type there is further exacerbation with loss of weight and lassitude There may be endocarditis associated with an acute disseminated lupus erythematosus when it is known as Libman-Sacks disease.

Diagnosis : (I) In the chronic discoid type there may be one or many circular or oval or butterfly-like lesions covered with adherent scales on erythematous base with a pigmented periphery associated with itching on the face, back and chest, (2) In the chronic disseminate or in acute disseminate types macular, urticarial or erythema multiforme type of lesions occur with inflammatory exacerbation of the facial lesions accompanied with malaise, irregular fever of unknown etiology and arthralgia and weakness.

(3) [illegible] in the chronic discoid [illegible] presence of *keratotic* plugs at the mouths of the hair-follicles. Acanthosis of the st. mucosum is seen with degeneration of the stratum basalis. In the dermis there is destruction of the elastic tissue and collagen fibres with dense *lymphocytic infiltration* which *runs* parallel with the dermo-epidermal junction. There is dilatation of the blood vessels and lymphatics. *In the acute* disseminate type there is hyperkeratosis, atrophy *of* stratum mucosum and degeneration of *the stratum* basalis. Dilatation of

the blood vessels and lymphatics with inflammation of the elastic and collagen fibers in the dermis with moderate lymphocytic infiltration, (4) Examination of Blood for total count of W B C, R B C, Differential count and Hæmoglobin per cent, (5) E S R—high, (6) blood W R is false positive, (7) Urine examination shows albuminuria, (8) L E cells and L E phenomenon are positive in acute lupus erythematosus only

Differential diagnosis (1) Seborrhoic dermatitis, (2) Lupus vulgaris, (3) Syphilis, (4) Alopecia areata and (5) Psoriasis

Prognosis Is favourable in chronic discoid type whereas in the chronic disseminate it is fair but the acute type ends in death in the tropics Alopecia is permanent

Treatment Prophylactic—for some people it is better to avoid the exposure to sun It is better to use a screening ointment while going out in the sun The ointment should consist of 1 per cent Para amino benzoic acid in vaseline Curative—(1) To investigate all the foci of infection and to treat them if found, (2) Bismuth—colloidal bismuth injections are given in doses of 0 2 gm biweekly for 10 weeks after urine examination Hygiene of mouth is important Orally bismuth may be given in dose of 75 mg of Bistrimate thrice daily, (3) Crude liver extract may be injected intramuscularly in dose of 2 c c biweekly, 12 such, (4) Gold sodium thio sulphate is given by intravenous injections starting with 5 mg and increasing gradually by 1 mg every week until 30 mg is given, (5) Vitamin B_{12} in dose of 500 microgram can be injected intramuscularly every day to allay itching, (6) Atebrine orally has been used in dose of 25 mg

3 times daily for the first week, 25 mg 2 times daily for 10 weeks or more. It is difficult to carry the atebrine treatment for its toxic effects such as yellow colouration of the skin, pain in the abdomen and atebrine psychosis.

Locally may be used—(1) 1 p. c. Quinine bihydrochlor. ointment, (2) 5 p.c. Para Amino Benzoic-Acid-ointment, (3) 2 p.c. Ung Hydrarg Ammon and (4) Lint. Calamine.

For subacute or acute disseminute types of cases (1) ACTH-aquous 40 units is injected intramuscularly twice daily for the first week, ACTH-gel 40 units is injected intramuscularly once daily for the second week. Follow-up treatment can be carried on by long-acting ACTH, (2) Cortisone may be given by injection or by the oral route in dose of 25 mg tablet every 6 hours for the first week and then in gradnated dose. Sodium salt is not given but Potassium salt such as K-salt (Calcutta Chemical) is allowed during the corticotropine and cortisone therapy, (4) Blood transfusion is helpful in the acute stage of the disease, (5) Vitamin C (500 mg), is injected intramuscularly twice daily, (6) Multi-vitamin is given in high dosage by mouth, (7) Penicillin crystallin 'G' in aquous solution in dose of O·5 Mu daily is to be injected for any intercurrent infection, (8) Sulpha-drugs are not used, (9) Quinine bisulphate may be given, (10) Oxygen inhalation is helpful.

SCLERODERMA

Definition : Is a skin disease characterized by the appearance of localized or generalized smooth, hard, white areas with a pigmented periphery on the skin which is red and swollen in the early stage later becoming atrophied and smooth.

Etiology: Varieties are found as (1) Localized type or morphœa and (1) Generalised type. Cause not known Acute infectious diseases, cancer, sunlight and psychological upsets may be responsible Women are commonly affected Found in the tropics. Common amongst young adults.

Signs and symptoms: Localized or diffused swelling of the skin with redness. Gradually these areas become smooth, whitish in colour, fixed to the underlying tissues and become atrophied. Site—may be on the face (Fig 174 & 175) trunk, upper and lower extremities



Fig. No 174
Scleroderma
(Morphea type on the forehead)

(Fig No 176). Commonly *seen on hands* which extends upwards and then on face. Morphea often occurs on the extremities, *face and the middle-line* of the forehead. Scleroderma *of fingers* is called



Fig. No 175
Morphea
(Scleroderma on forehead)

sclerodactyle (Fig No. 176 & 177) It is non-itchy and not tender Calcium deposition in different tissues is called *Calcinosis cutis* or Weissenbach syndrome.



Fig No. 176
Generalized scleroderma with sclrodactyle
(Case of Dr S. C Desai)

Diagnosis : (1) Localized or generalized inflammed skin or atrophied skin, (2) Sites—forehead or extremities or trunk, (3) Biopsy—histopathology shows flattening of the dermo-epidermal junction *with atrophy* of the *hair*-follicles and sweat-glands. *Degeneration*



Fig No 177
Scleroderma with sclerodactyle
(Case of Dr S C Desai)

and homogenization of the collagen fibers with the loss of parallel arrangement of the elastic fibers (Fig No 178), (4) Blood calcium level is high (5) Creatine estimation is high (6) 17 Ketosteroid—low urinary Ketosteroid level

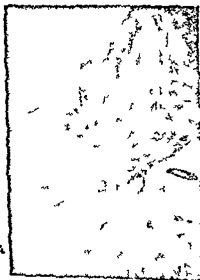


Fig No 178
Scleroderma
Histopathology

Differential diagnosis (1) Leucoderma (2) *Psoriasis*

Prognosis Is fair in morphea as it may be cured even without treatment In generalized type patient dies of some intercurrent disease

Treatment : Prophylaxis—nothing is known.

Curative—(1) Physical therapy—such as massage, application of heat, (2) Para-Amino-Benzoic-Acid in dose of 12 gram daily by mouth for about 12 weeks, (3) Thyroid extract by mouth gr½ daily, (4) Ditachysterol in dose of m. 15 thrice daily by mouth, (5) Niacin and neostigmine 10 mg 2 to 3 times a day are helpful, (6) Penicillin injection is helpful, (7) Vitamin B-complex in high dose is beneficial, (8) Cortisone can be given, (9) ACTH may also be injected during acute exaccerbations. Sympathectomy is sometimes helpful, (4) Glyceryl Trinitrate Ointment may be used locally.

DERMATOMYOSITIS

Definition : Is an acute or chronic skin disease characterized by inflammation of muscles and skin

Etiology : Is unknown. Sometimes coccal infection may be responsible. Chronic systemic disease and visceral cancer may be the precipitating causes

Age—at any age. Sex—both sexes

Signs and symptoms : Weakness, pain and tenderness in muscles are common Muscles of the extremities are often first affected symmetrically (Fig. No. 179). In the early stage muscles are tender and normal in

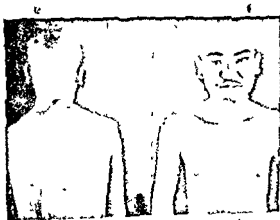


Fig. No. 179
Dermatomysitis
(Case of Dr. S. C. Desai)

shape but later on the muscles become hardened Contracture may result Respiration and swallowing may be very difficult Enlargement of spleen and lymphatic glands may be associated There are reticulated erythematous plaques on the trunk, extremities and on the dorsum of the hands

Diagnosis (1) Progressive weakness in muscles resulting in hardening of muscles and difficulty in respiration and swallowing (2) E S R—high, (3) Blood—serum estimation shows high globulin, (4) Urine analysis shows creatinuria (5) Biopsy—histopathology shows inflammation of muscle fibers and infiltration with polymorphonuclear cells Proliferation of connective tissues and hyalinization of collagen fibers in the dermis

Differential diagnosis (1) Lupus erythematosus and (2) Scleroderma

Prognosis Death may result in few weeks in acute but when recovery occurs there is muscular weakness left In chonic cases patients suffer long and the disease may become stationary

Treatment Prophylaxis—is not known Curative (1) Removal of infective foci (2) Auto vaccine therapy may be helpful and non specific protein therapy with milk injection may be tried (3) Penicillin therapy is sometimes indicated (4) Vitamin E gives some beneficial effect (4) Cortisone—has been found to be helpful in some cases (5) ACTH is also helpful, (6) Androgen therapy has been rarely helpful (7) Antihistamin drug may be used (8) Blood transfussion is advocated (9) Rest in bed is essential (10) Diet—high protein diet should be given consisting of meat fish egg and for the vegetarians milk and channa

CHAPTER XIV

SEBORRHOEIC DISORDERS OF THE SKIN

Seborrhoeic disorders include: (1) Acne vulgaris, (2) Acne rosacea and (3) Seborrhoea.

ACNE VULGARIS

Definition: Acne vulgaris is a chronic inflammation of the pilo-sebaceous follicle accompained with comedone formation and seborrhoea particularly of the face during adolescence.

Etiology: (1) Endocrine: seen commonly in adolescents and increases during menstruation. Is supposed to be due to the imbalance of oestrogen and androgen. Predominence of androgen has been observed in acne vulgaris. Sex glands—when there is an excessive production of androgen upsetting the endocrine balance with estrogen it results in the production of acne vulgaris. There is an exacerbation in women during menstruation, (2) Metabolic disturbance—as in thyroid deficiency, (3) Hypovitaminosis A and C—low vitamin intake causes acne vulgaris, (4) Diet—in some patients particular diet causes aggravation of acne such as carbohydrate-rich and fatty food, (5) Sex—found in both sexes, (6) Age—common at the adolescent age but may be found in children and also in women during pregnancy. Common age is 13 to 30 years.

Signs and symptoms: Lesions are mainly papules, nodules and pustules situated on the face

(Fig No 180) shoulders and chest There may be inflammation present with the papular type but



Fig No 180
Acne vulgaris
(showing black heads
al over the back)

nodular types are always accompanied with inflammation The pustular lesions of acne vulgaris are said to be due to the entrace of pyogenic organisms in the hair follicles The important lesion in acne vulgaris is the formation of comedo caused by the formation of excessive keratinization at the mouth of the hair follicle The acne may be cyst like when it is called *cystic acne* (Fig No 181) Scalp shows excessive amount of oily secretion called seborrhoea Sometimes the face is also oily A type of acne has been described as the *tropical acne* Tropical acne is seen only in foreigners visiting tropics and is characterised by the exacerbation of the ordinary acne vulgaris



Fig No 181
Acne cystica
(Case of
Dr B S Verma)

Diagnosis (1) Age of patient —adolescent age (2) Papular or pustular eruption with black heads on face, back, chest and shoulders

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Fig No 181
Acne cystica
(Case of
Dr B S Verma)

Diagnosis (1) Age of patient —adolescent age, (2) Papular or pustular eruption with black heads on face, back, chest and shoulders

with inflammation and oiliness of the face and scalp (3) Histopathology—perifolliculitis with follicular plug of sebum

Differential diagnosis (1) Acne rosacea, (2) Oil acne, (3) Tar acne, (4) Acne due to bromide and iodide and (5) Pustular syphiloderm

Prognosis Acne vulgaris troubles a person between the age of 13 to 30 years Prognosis is good in acne vulgaris but in acne cystica sinuses, scars and keldoids usually develop.

Treatment (1) Diet is most important Normal diet is given including meat, fish, milk, vegetable rice and bread Fatty food is cut down to the minimum Carbohydrate intake should be restricted (2) Exercise—walking, swimming, joining outdoor games are essential Sedentary habit is bad in acne patients (3) Locally—Bland soap should be used regularly and particularly face should be washed before going to bed The following lotion may be applied every night in mild cases of acne vulgaris (1) Zinc Sulph—gr 25 *Potassium* Sulphuratum—gr 15 Aqua Rosae—dr 1, Aqua distilata—oz 1. Ft lotio To apply every night (2) An ointment may be rubbed every night after washing the face with a bland soap and warm water The rubbing of the following ointment should be done for 3 nights followed by rest for one night and repeating 6 times Resorcin—gr 5 Acid Salicylic—gr, 10 *Vaseline alba—oz 1 or Sulpher ppt*—gr 10, Acid Salicylic—gr 10, Vaselin alba—oz 1

(c) X ray therapy or ultra violet therapy may some times be helpful, (4) Staphylococcal toxoid vaccine

may be given in pustular acne in gradually increasing doses, (5) Antibiotic—penicillin crystalline 'G' is often helpful in pustular acne vulgaris, (6) Shampooing of the scalp is helpful with Selsun (Abbott) when there is seborrhoea present, (7) Endocrine therapy—some patients do well with oestrogen therapy In females it is given for 14 days starting 14 days before the expected menstruation and the drug should be stopped with the appearance of menstruation Oestrogen may also be carefully given even to the males and the patient must be watched for the development of gynecomastia Thyroid in small doses (gr ½ daily) is sometimes helpful in acne vulgaris, (8) Vitamin therapy—Vitamin A in high doses (Arovit-Roche 100,000 I U) daily for a period of 6 months with vitamin C are helpful, (9) Anaemia when associated in the tropics should be treated with iron therapy and constipation must be corrected, (10) Acne scars may be treated by Carbon dioxide snow application

ACNE ROSACEA

Definition Is a chronic inflammation of the pilosebaceous follicles of the center of the face

Etiology (1) Gastrointestinal troubles are commonly associated, (2) Psychological maladjustment may be responsible, (3) Vitamin-B_2 deficiency may be found

Signs and symptoms Redness of the center of the face with hot curries, strong and hot tea, coffee, alcohol and after gastro intestinal upset and during menstruation are the early signs Later on the redness becomes permanent with papules and pustules on the chin, nose, malor regions and on the forehead

with inflammation and oiliness of the face and scalp, (3) *Histopathology—perifolliculitis with follicular plug* of sebum.

Differential diagnosis: (1) Acne rosacea, (2) Oil acne, (3) Tar acne, (4) Acne due to bromide and iodide, and (5) Pustular syphiloderm.

Prognosis: Acne vulgaris troubles a person between the age of 13 to 30 years. Prognosis is good in acne vulgaris but in acne cystica sinuses, scars and keldoids usually develop.

Treatment: (1) Diet is most important. Normal diet is given including meat, fish, milk, vegetable, rice and bread. Fatty food is cut down to the minimum. Carbohydrate intake should be restricted, (2) Exercise—walking, swimming, joining outdoor games are essential. Sedentary habit is bad in acne patients, (3) Locally—Bland soap should be used regularly and particularly face should be washed before going to bed. The following lotion may be applied every night in mild cases of acne vulgaris: (1) Zinc Sulph—gr. 2·5, Potassium Sulphuratum—gr. 15, Aqua Rosae—dr. 1, Aqua distilata—oz. 1. Ft lotio. To apply every night. (2) An ointment may be rubbed every night after washing the face with a bland soap and warm water. The rubbing of the following ointment should be done for 3 nights followed by rest for one night and repeating 6 times: Resorcin—gr. 5, Acid Salicylic—gr, 10, Vaseline alba—oz. 1. or Sulpher ppt.—gr 10, Acid Salicylic—gr. 10, Vaselin alba—oz. 1.

(c) X-ray therapy or ultra-violet therapy may sometimes be helpful, (4) Staphylococcal toxoid vaccine

may be given in pustular acne in gradually increasing doses, (5) Antibiotic—penicillin crystalline 'G' is often helpful in pustular acne vulgaris, (6) Shampooing of the scalp is helpful with Selsun (Abbott) when there is seborrhoea present, (7) Endocrine therapy—some patients do well with oestrogen therapy. In females it is given for 14 days starting 14 days before the expected menstruation and the drug should be stopped with the appearance of menstruation. Oestrogen may also be carefully given even to the males and the patient must be watched for the development of gynecomastia. Thyroid in small doses (gr. ½ daily) is sometimes helpful in acne vulgaris, (8) Vitamin therapy—Vitamin A in high doses (Arovit-Roche-100,000 I.U.) *daily for* a period of 6 months with vitamin C are helpful, (9) Anaemia when associated in the tropics should be treated with iron therapy and constipation must be corrected, (10) Acne scars may be treated by Carbon dioxide snow application

ACNE ROSACEA

Definition Is a chronic inflammation of the pilo-sebaceous follicles of the center of the face.

Etiology (1) Gastrointestinal troubles are commonly associated, (2) Psychological maladjustment may be responsible, (3) Vitamin-B_2 deficiency may be found.

Signs and symptoms Redness of the center of the face with hot curries, strong and hot tea, coffee alcohol and after gastro intestinal upset and during menstruation are the early signs. Later on the *redness* becomes permanent with papules and pustules on the chin, nose, malor regions and on the forehead

Gradually the tip of the nose becomes enlarged and is called *Rhinophyma.*

Diagnosis : (1) History of chronic gastro-intestinal upset or psychological troubles, (2) Flushing of the face after hot food or during menstruation, (3) Papules and pustules on the centre of the face, (4) Riboflavin deficiency in the eyes, mouth etc. (5) Histopathology shows dilatation of the dermic vessels and infiltration of the pilosebaceous follicles with small round cells, (b) Gastric analysis shows achlorhydria or hyperchlorhydria.

Differential diagnosis : (1) Acne vulgaris, (2) Drug rash, (3) Secondary syphilis, (4) Tuberculosis cutis.

Prognosis : Fair with treatment.

Treatment : Diet should be regulated and gastro-intestinal troubles should be treated with Riboflavin injection and Vitamin B-complex orally. Acid Hydro-chlor dil in doses of m. 10 soon after food is helpful. If a psychological trouble is causing the disease some sedative is given such as phenobarb gr. ½ at night for 3 to 4 nights or bromide in mixture with valerian for several nights. Psychotherapy is helpful.

Locally lotio Calamine is useful. Sometimes 1 p.c. Resorcin ointment rubbing helps. As a routine Ung Sulpho-salicylic is rubbed twice daily. Superficial X-ray therapy is also indicated. Rhinophyma is treated by plastic surgery.

SEBORRHOEA

Definition : Is a constitutional recurrent skin disease characterised by inflammation and scaling which

starts from the scalp and spreads down the face, neck, trunk and the limbs

Etiology It is much less prevalent in the tropics Sebaceous saretion is influenced by emotional factors, digestive disturbances infections nutritional deficiencies and by external causes such as chemical The disturbance in the sebaceous secretion produces the seborrhoeic condition

Classification (1) Seborrhoic capitis is the sebor hoea of the scalp and (2) Seborrhoeic dermatitis is the seborrhoea of the trunk May occur at any age but commonly at puberty and affects both sexes

Signs and symptoms In children blackish crusts are found on scalp and behind the ears and groins Scaly eczematous lesions may involve the face and scalp (Fig No 182) in an adult which is rarely seen in an infant Flexure surfaces are commonly involved



Fig No 182
Seborrhoea

such as as the axilla and crural regions. The scalp, the forehead, eyelids, ala nasi, meatus of the ear, face, even lips may be involved as also the axillæ, intrascapular regions, front of the chest *down to the pubic* region. Even palms and soles may develop pompholyx type of lesion in seborrhoea. Genitalia may be affected in both sexes with seborrhoea. There may be oozing, crusting or only dry scaling. Sometimes erythematous patches may be seen. The seborrhoic lesions are itchy. Blepharitis, melanoderma, alopecia, seborrhoic wart in old age may develop as complications.

Diagnosis: (1) Itching, oozing or scaling dermatitis, (2) Typical distribution, (3) Familial history, (4) Histopathology shows hyperkeratosis and parakeratosis of the stratum cornenm. There is acanthosis and intra-cellular edema in the stratum mucosum. Dermis shows dilatation of the papillary vessel with perivascular lymphocytic infiltration, (5) Blood sugar may be high normal or may be often hyperglycaemic.

Differential diagnosis (1) Psoriasis of the scalp is differenciated by matting of the *hair with sebum in* seborrhoic capitis but histopathologically there is no difference, (2) Eczema of scalp is difficult to diagnose histopathologically but in seborrhoea there is typical seborrhoic distribution if it spreads down the trunk, (3) Ringworm of the scalp is diagnosed by microscopical examination of the infected hair, (4) Infantile Eczema—generally starts on the cheeks as grouped vesicles which soon starts oozing whereas the seborrhoea starts on the scalp which extends down the face, neck and trunk, (5) Pityriasis versicolor by the microscopical examination where microsporon *furfur*

is found (6) Dermal Leishmaniasis in the depigmented stage by the history of having had Kala Azar and by the histopathological examination of the skin for Leishman—Donovon bodies, (7) Pityriasis rosea is differential by its seasonal occurrence and typical bathing trunk distribution (Genji and underwear) and pink oval lesions along the clevage line of the ribs with scales at the periphery looking towards the centre

Prognosis Is good with treatment but is never cured

Treatment General patient should avoid sedentary habit and should take regular exercise Bowels must be kept moving Worry and anxiety must be avoided as far as possible Diet should be mixed type with vegetables Milk should be avoided Alkali is given to keep urine alkaline in the acute stage Vitamins are of particular value like Vitamin B complex Vitamin A and Vitamin C Sedative is required in the acute stage such as pheno barb gr ½ twice daily Methionine (Neomethidine—Neo Phama) is helpful

Locally a shampoo should be used for the scalp like Selsun (Abbott) or a shampoo containing Liq Picis Carb det—dr 1 Oil Recini—dr 1, Liquid soap—oz 2 After washing scalp with shampoo a lotion may be used for the scalp containing Resorcin—gr 2 Liq Picis carb det—m 4 50 p c Spirit Rect—ad oz 1 For the scalp an oil free shampoo is used 2 to 3 times a week followed by the application either of lotion or ointment Acid Salicylic—gr 10 Sulpher ppt—gr 10, Oil of cade—m 10 Vaseline alba—oz 1 ung for external use Locally in the acute stage for the body Argenti Nitras—gr 5 Aqua destil—oz 1 Ft lotio supply oz 8 in a coloured phial To apply every hour

for one day followed by on the 2nd or 3rd day a lotion containg : Sulpher ppt.—gr. 10, Calamine ppt—dr. 1. Aqua Distil—oz 10.

In the chronic stage an ointment is used for the body. Sulpher ppt.—gr 10, Acid Salicylic—gr. 10, Vaseline alba - oz. 1. Ft. Unguentum. Use ointment twice daily *for the body or scalp after bath with bland soap.*

PSORIASIS

Definition : is a chronic noninfectious skin disease characterised by silvery scales on erythematous base with itching and distributed mainly round the elbow, round the knee, sacral region and on the scalp.

Etiology : Is not known yet. It is quite a common disease in the tropics. It forms about 3 per cent of all skin cases in India. It affects both sexes. Cases of psoriasis have been found in patients between the age of 3 months to 90 years but is commonly found in adults. Metabolic disorders are sometimes held responsible for its development. Endocrine disorders, vitamin C deficiency with higher vitaminosis A, stress and strain and psychosomatic factors are said to be responsible. Septic focii are responsible predisposing factors and has often preceeded an eruption of psoriatic lesion. Sometimes psoriasis and arthritis are associated. Climate may have some influence and psoriasis cases are commonly seen in winter months in the tropics.

Signs and symptoms : Lesion of psoriasis may start as a macule which is reddish in colour. Silvery white scales appear on the macule. Macules may become papules after a time. When a macule or a papule is scraped

by a pointed instrument scales on either side of the line appear and this test is called *tache de bouge* On scraping bleeding points appear Distribution is on the extensor sufaces Lesions may be found on the scalp on the trunk, elbows (Fig No 183) sacral and gluteal



Fig No 183
Psoriasis forearm
(Case of Major N R Gupta)



Fig No 184
Psoriasis
(Acute aczematous type)

regions knees (Fig No 184) umbilicus and on the nails of fingers and toes Psoriatic lesions may be found on palm and sole also and is known as palmer or planter psoriasis or pustular psoriasis Lesions may develop along the line of a scratch on the skin which is called *Koebner's phenomenon*

Varieties of psoriasis (1) Psoriasis punctata lesions look like small points (2) Psoriasis guttata lesions look like rain drops (Fig No 185), (3) Psoriasis nummularis lesions look like coins, (Fig No 186)

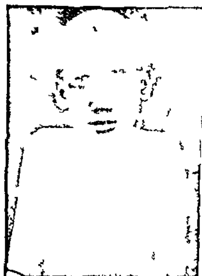


Fig No 185
Psoriasis
Guttate type
(Case of Dr R P Gupta)



Fig No 186
Psoriasis
(Case of Dr K C Sahu)

(4) Psoriasis circinata lesions are in patches and form polycyclic figures, (5) Posiasis figarata lesions are like figures, (6) Psoriasis follicularis lesions affecting hair follicles, (7) Psoriasis arthropathica-lesions are associated with arthritis, (8) Pustular psor asis-erythemato-pustular lesions appear on the instep of sole and on the palm, (9) Eczematous psoriasis, (10) Psoriasis seborrhoea and (11) Flexural psoriasis

Diagnosis (1) Typical lesion silvery scales on erythematous base distributed on scalp, extensor surfaces, elbows, knees and sacral regions and also on the nail, (2) Tache de Bouge test positive, (3) Koebner's phenomenon positive, (4) Biopsy and histopathology-Hyperkeratosis, parakeratosis, elongation of the rete pegs, absence of stratum granulosum, thinning of rete mucosum above the papillae, oedema of the papillary body and the presence of microabscesses of Munro

Differential diagnosis (1) Eczema, (3) Seborrhoic dermatitis, (3) Squamous syphilis, (4) Pityriasis rosea, (5) Lichen planus (6) Lichen simplex chronicus (Widal), (7) Tinea corporis, (8) Acrodermatitis perstans, Acrodermatitis tropicalis and pompholyx should be differentiated from pustular psoriasis

Prognosis Condition can be improved with treatment and patient may be made temporarily free but permanent cure is not possible Relapses are common

Treatment General (1) Any systemic disease should be treated and septic foci should be *removed*, (2) Low protein diet is advisable Vegeteranian diet with milk is commonly recommended Vitamin C rich and low vitamin A diet is helpful, (3) *Internally*-arsenic

by mouth such as Fowler's solution in dose of one drop thrice daily for a long time and the dose is often increased gradually, (4) Vitamin C in high dose (500 mg. I. M. twice daily) during the acute stage and in small dose (500 mg. orally daily) are advocated during the chronic stage. Other vitamins such as Vitamin A and Vitamin D are sometimes helpful. ACTH in high dose during the acute stage and in small dose for the follow up is a helpful adjuvant to the therapy but ACTH is never a specific for psoriasis. There is frequently an exacerbation in psoriasis after this therapy but is only helpful in prosiasis arthropathica. Cortisone should never be given in psoriasis because when it is stopped the relapse is very severe.

Locally-during the acute stage 1 p. c. Ung. Ichthyol (Ichthyol-Gr. 5 in Vaseline alba oz. 1). During the subacute stage Ung. Hydrarg ammon (Hydrarg. ammoniata gr. 20 in Vasiline oz. 1) alone or with 2 p. c. Acid Salicylic are used.

During the chronic stage 2 p.c. Ung. Chrysarobin gradually increasing to 5 p.c. for lesions localised on body except face and scalp is used otherwise irritation may give rise to acute conjunctivitis For the scalp 2 to 5 p c. oil of cade in vaseline together with 2 p.c. acid salicylic.

For ambulatory patients painting with crude coal tar is very helpful but should not be applied long as it is one of the carcinogenics.

Electrotherapy-generalised ultra-violet exposure.

Superficial X-ray therapy—In generalised eruption 40r with 75 KV is valuab[le] but for small isolated patches 80r with 75 KV is r

psoriasis and for the psoriasis of nails 6 to 8 weekly exposures of superficial X ray therapy in dose of 80 r with 75 KV are given Thorium X solution (1,500 Electrostatic unit per ml of solution) may bs locally applied on chronic lesions anywhere on the body.

LICHEN PLANUS

Definition Is a chronic skin disease characterized by reddish brown papules or blackish-red hyperkeratotic plaques on the skin of the front of the wrist and other parts of the body including the mucous membranes of mouth, tongue, glans penis and vagina

Etiology It is not an infectious disease Both sexes are affected equally Age—common in adults but no age is exempt No cause is known but worry seems to play some part in its development It forms about 2 p c. of all the skin cases in the tropics

Signs and symptoms May start with itching pin-point shiny lesions on the skin or as one or two very small polygonal papule reflecting light infront of both wrists or both shins Distribution is symmetrical over the flexor surfaces forearms wrists (Fig. No 187), shins, ankles (Fig No 188) but sometimes seen only on one side of the body The polygonal papules may coalesce and form a big sheet on the trunk when it is called *Lichen pl nus dissen inatus* (Fig No 189) when papules become very prominent and form a plaque commonly on the front of the shins it is known as (Fig No. 188) *Lichen planns hypertrophicus* Rarely seen like a band on one side of the body and is called *Lichen linearis* Sometimes whitish oval spots are seen called

Lichen planus atrophicus. These spots are conglomeration of polygonal atrophic macular shiny areas. When seen with a lens a dimple may be seen on each of the papules and it represents the opening of the hair follicle. Fine white points and white lines



Fig No 187
Lichen Planus

may be seen on the lesions with a magnifying glass and are known as *Wickham's striæ*. Lichen planus

of skin heals after leaving pigmentation but occasionally white atrophic-looking spots are left. These black or



Fig. No. 188
Lichen planus
hypertrophicus
(Case of Dr. N. C. Sanyal)



Fig. No. 189
Lichen planus
disseminatus

white spots disappear ultimately. Rarely lichen planus affects one of the limbs as a band such as the inferior extremity from heel to the groin. This is a slowly forming skin disease taking several months to develop with itching and typical Koebner's phenomenon.

Diagnosis : (1) Polygonal flat-topped itching papules which are shiny and *situated* on the front of the wrist, forearm, *ankle* and leg and are symmetrical

Lichen planus atrophicus. These spots are conglomeration of polygonal atrophic macular shiny areas. When seen with a lens a dimple may be seen on each of the papules and it represents the opening of the hair follicle. Fine white points and white lines



Fig No 187

Lichen Planus

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Fig No 188
Lichen planus
hypertrophicus
(Case of Dr N C Sanyal)



Fig No 189
Lichen planus
disseminatus

white spots disappear ultimately Rarely lichen planus affects one of the limbs as a band such as the inferior extremity from heel to the groin This is a slowly forming skin disease taking several months to develop with itching and typical Koebner s phenomenon

Diagnosis (1) Polygonal flat-topped itching papules which are shiny and situated on the front of the wrist, forearm, ankle and leg and are symmetrical

in distribution and may be present on the mucous membrane of the mouth as white patchy lesions, (2) Lesions may develop on a scratch—positive Kœbner's phenomenon, (3) Histopathology—Hyperkeratosis, thickening of the stratum granulosum and ,acanthosis. Degeneration of the *dermo-epidermal* junction. Round cell infiltration in the dermis.

Differential diagnosis: (1) Psoriasis, (2) Pityriasis rosea, (3) Discoid lupus erythematosus, (4) Papular syphiloderma, (5) Striate lichen planus from lichen striatus.

	Striate Lichen planus	Lichen Striatus
1. Koebner's phenomenon	present	absent
2. Itching	present	absent
3. Histopathology	like lichen planus	like eczema

Prognosis: Is a chronic skin disease. Generally it is completly cured but may relapse

Treatment: General treatment—Improvement of general health. Psychosomatic troubles should be taken into account and change of surroundings is advisable Tonic containing vitamin B complex is valuable

Internally Arsenic (Stovarsol—25 mg. tablet), vitamin B-Complex and Enesol (Comar cie) injected I M., Bismuth (0·2 gm.) is given by intramuscular injection and can be repeated after a month's interval. Mercury as Hydrag perchlor-gr 1/16 per dose, three times daily orally and locally 4 p c. Ung Hydrag. Ammon are used as a routine. Treatment of lichen planus is mercury in and mercury out. X-ray therapy locally is valuable. In a localised lesion 80 r weekly with 75 KV is given for 4 to 6 times at weekly intervals

but in gensralised or in acute cases 40 r at 95 KV in one or two doses are recommended

PITYRIASIS ROSEA

Definition Is an acute skin disease characterised by erythematosquamous patchy lesions distributed on the trunk, arms and thighs with slight itching

Etiology It is a mildly infectious acute skin disease Cause is not known Virus, internal toxin and treponema may be responsible Quite common in the tropics and forms about 0 1 p c of skin cases It is seasional Seen at the chage of seasions Affects all ages and both sexes

Signs and symptoms An Erythemato-squamous circular lesion about an inches in diameter which appears on the pectoral, scapular, umbilical or inguinal regions This patch is not itchy and is called the *herald patch* Herald patch is present in one third of all cases of pityriasis rosea 7 to 8 days after the appearance of the herald patch small oval reddish rash appear on the body while the herald patch gets almost cleared up by that time The lesions are papular red spots becoming oval reddish patches of one fourth inch in size. Scales are arranged at the periphery which look towards the centre As the lesions grow old the reddish colour changes into brownish red to brown and ultimately when healed look like depigmented spots Seen over the trunk and symmetrically down the arms upto the elbow and down the thighs as far as the knees but may be generalized also. Face is not affected The rash persists for only six weeks and is slightly itchy Sometimes sore throat preceeds the rash

Diagnosis : (1) Seasional appearance, (2) Typical symmetrical distribution and on the areas covered by the underwears (Genji and underpant), (3) Rose red colour of lesions with scales arranged at the periphery and lesions arranged along the clevage line of the ribs, (4) *Biopsy* and histopathological examination shows—parakeratosis and acanthosis, oedema in the dermis and migration of cells in the epidermis.

Differential diagnosis : (1) Ringworm, (2) Pityriasis versicolor, (3) *Psoriasis*, (4) Seborrhoic dermatitis, (5) Macular syphilide, (5) Leprosy, (6) Dermal leishmaniasis.

Prognosis : Good even without any treatment.

Treatment : No treatment is usually necessary. Lotio Condy's (Pot. Permanganate gr. 20 in Aqua oz. 1) bath daily followed by 2 p.c. Ung Acid Salicylic. Sometimes generalised ultra-violet therapy clears all the lesions by only one exposure. Antibiotic like aureomycin, terramycin, chloromycetin, Ilotycin may be used for 4 days orally with vitamin B-complex.

AINHAM

It also known as *Dactylolysis spontanea*.

Definition : Is a chronic skin disease occurring specially in the tropics and is characterized by the formation of a fibrous ring around the little toe of one or both feet. The ring gradually narrows and in several weeks to several years the toe falls off.

Etiology : Is unknown. Specially seen in the tropics.

Types—(1) Ainham and (2) Pseudo-ainham.

Signs and symptoms A groove generally appears on the side or dorsum of one of the little toes (Fig No 190) which as it deepens it also encircles the toe and

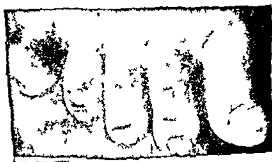


Fig No 190
Ainham

the time taken may be several weeks The depression is formed by a fibrous ring which gradually narrows and cuts the toe without pain or bleeding The underlying bone also gets thinned but there is no osteomyelitic change or rarefaction seen Sometimes it effects the little toe of the other foot (Fig No 191) in

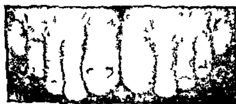


Fig No 191
Ainham

several weeks to several years after the affection of the former toe Rarely the third and even the fourth

toe of the other foot also is affected. There is no discharge or signs of inflammation below the condition. Histoperthology of the fibrous ring shows thickening of all the layers of the epidermis (Fig. No 192). There



Fig. No 192
Histopathology of Ainham
(Biopsy of the fibrous constriction)

is no systemic reaction. The tarsal bone gives way and the toe falls off Radiologically there is thinning of the tarsal bone (Fig. No 193). Pseudoainham is either congenitally absent toe, finger or limb or is due to other diseases like leprosy, scleroderma.

Differential diagnosis · (1) Leprosy, (2) Chronic ulcer.

Treatment Prophylaxis—to use foot wear

Curative—cutting the bands at different places

Cauterising with Iodine Penicillin injection for 2 weeks Warm condy s bath

POROKERATOSIS (MIBELLI)

Definition Is a chronic disease characterised by keratotic ring shaped lesions occuring on the body

Etiology No cause is known It is a hereditary keratodyskeratosis It is supposed to be due to vitamin A deficiency Is found in the tropics Men are commonly affected Age starts generally at the prepubertal age and stays throughout life Forms 0 1 p c of skin cases



Fig No 193
Sklagram of left foot with Ainham of toe
(Narrowing of bone of the little toe)

Signs and symptoms Sometimes it is itchy Usually there is no symptom at all The lesions may be very few in number (Fig No 194) or starts insidiously over a sweat duct opening hence called ' poro as a keratotic follicle or keratotic papule This papule enlarges in size and gets

depressed in the centre until a keratotic ring is formed and is described as kerato-atrophoderma. In size the largest



Fig. No 194
Porokeratosis (Mibelli)
(Lesions on latrel aspect of the right Knee joint)

one may be half an inch in diameter with a black keratotic ring enclosing an atrophied skin. The extension is centrifugal. The hair, sweat and sebaceous glands on the atrophied skin undergo atrophy. Lesions are multiple but rarely single May occur all over the body on the skin and rarely on the mucous membrane.

Diagnosis: (1) The typical keratotic ring-like balckish lesions, (2) Sites on the dorsum of hands, buttocks, thighs are common places, (3) Biopsy—histopathology shows hyperkeratosis of all the layers of the epidermis. Corps ronds are found in the epidermis. In the dermis there is atrophy of the glands (Fig. No. 195).

Differential diagnosis: (1) Basal-cell carcinoma and (2) Senile keratosis.

Prognosis Is not curable

Treatment Massive vitamin A therapy (Arovit-Roche tablet 100,000 i u) is given by mouth for a long time Excision of the lesions may be done



Fig No 195
Porokratosis (Mibelli)
Histopathology

MILIARIA RUBRA

This is commonly known as prickly heat

Definition Is a tropical skin disease characterized by the formation of erythematopapular rash with pruritus

Etiology High humidity with tropical heat is supposed to be responsible for the causation of prickly heat Low vitamin A nutrition is responsible. Due to the maceration of the stratum corneum the sweat ducts get blocked and thus the rash is produced from each of the blocked sweat duct Common amongst young adults but may be found in any age and in both sexes

Signs and symptoms. Erythemato-papular rash found during summer and monsoon later becoming papulo-vesicular and some becoming pustular. Site-back, chest and flexure surfaces and sometimes the face is also affected but palm and sole are never affected. Covered areas liable to friction are generally affected. It is very pruritic When the sweat is retained and causes the dilatation of the ducts half pea sized multiple lesions particularly appear on the face which are known as *summer boils.*

Diagnosis : (1) Erythemato papulo-vesicular lesions on the covered areas of the body during the summer and monsoon months in a tropical country, (2) Itchy rash becoming squamous when the condition subsides leaving no scar, (3) Pilosebaceous follicles are free whereas the sweat ducts are involved, (4) Histopathology—blockage of the sweat duct with a keratotic plug. Intraepidermal part of the sweat duct is destroyed. Dermis shows edema with small round cell infiltration and dilatation of papillary vessels

Differential diagnosis (2) Sudamina, (2) Seborrhoea.

Prognosis Good.

Treatment. Prophylaxis is to avoid hard labour during the day in summer and to use electric fans Daily bath with cold water twice daily. Use of loose garments is a good prophylaxis in the tropics Hot tea and pickles in the food should be avoided. Vitamin A should be taken in dose of 50,000 i u twice daily

Curative—Application of lotio calamine is helpful. In the generalized type Vitamin A internally is helpful

CHAPTER XVI

METABOLIC DERMATOSIS

There are some skin diseases which are due to the disturbance in the mineral metabolism such as Calcium, Magnesium, Potassium Sodium in the body Calcium is most important as it is used in high dose in the form of Calciferol in the treatment of tuberculous infection of the skin Disturbance in protein metabolism has been observed in several diseases such as pemphigus and dermatomyositis Disturbance in lipid metabolism in psoriasis, carbohydrate metabolism in diabetes mellitus and prophyrin in porphyria

CALCINOSIS CUTIS

Calcium deposition in the body may be (1) Localised, (2) Generalised and (3) Metastatic

Local deposition of calcium in the skin is found secondary to various diseases such as scleroderma, milia, sebaceous cysts Generalised calcinosis cutis is supposed to result from circulatory insufficiency The metastatic calcinosis cutis is due to the hyperparathyroidism and is due to excessive intake of Vitamin D Ulceration may develop to excrete the calcarius material Normal blood calcium level is from 9 to 11 mg per 100 ml blood Normal blood calcium level does not signify normal calcium metabolism

Prognosis Is good in the localised and metastatic types whereas the generalised type is fatal

Treatment : There is no treatment for the generalised calcinosis cutis. In the localised type excision of the plaque with cutting down the intake of calcium and to stop the calcium therapy. Excising the plaque is also advocated.

PORPHYRIA

It is also known as porphyria cutanea tarda.

Definition : Is a chronic skin disease due to the defect in the porphyrin metabolism

Etiology : It is found in the tropics but is not a common ailment. It is due to an error of porphyrin metabolism. Affects all ages and both sexes. Types are (1) Congenital, (2) Acquired and (3) Secondary.

Signs and symptoms : In the congenital type the skin shows photosensitivity to light. Bullous rash is found on the face and exposed parts of the body. Lesions become eczematous and heals leaving pigmentation

When affects the skin of infants and children it is the congenital type but when the skin of the adults only are affected it is another variety of congenital type known as *Prophyria Cutanea Tarda* In this adult type the patient is commonly a female with hirsuitism. The patient may have intermittent abdominal pain.

Diagnosis : (1) Vesico-bullous or eczematous skin lesion on the face and exposed parts of the body, (2) Porphyrin estimation in fresh urine with Ehrlich aldehyde test—the urine shows red colour due to the formation of porphobilinogen, (3) Spectroscopic

examination of the urine shows uroporphyrin spectrum, (4) Histopathology shows edema of the collagen fibers with an intraepidermal bulla

Differential diagnosis (1) Epidermolysis bullosum and (2) Drug rash

Prognosis Is never cured

Treatment Protection of the liver with methionine (Neo methidine) Sometimes splenectomy is advocated

MYXEDEMA

Definition Myxedema is a metabolic skin disease due to the deficiency of thyroid hormone and is characterized by non pitting edema of the skin which is dry, rough waxy and with slow body movements

Etiology It is caused by the lack of thyroid function especially after thyroidectomy Occurs after the age of 40 years and is generally seen in women

Signs and symptoms Skin has a waxy feel and is dry and rough with nonpitting edema Lips become swollen and everted Swelling of the dorsum of hands and on the pretibial regions *pretibial myxedema* is a rare complication Hair becomes dry lustreless and falls from the scalp and eyebrows Baldness develops and there is absence of hairs on the lateral half of eye brows Nails become discoloured and are cracked Teeth become carious Pads of fat appear on the shoulders *Expressionless face and slow movements* are characteristics Anaemia is associated.

Prognosis Good

Treatment ; Thyroid extract gr. ¼ by mouth twice daily should be continued for a long time Cortisone therapy may be given with the restriction of table salt Hyaluronidase may be injected locally in pretibial myxaedema.

DISORDER OF CARBOHYDRATE METABOLISM

Diabetes Mellitus

The skin disorders due to the disturbance in the carbohydrate metabolism are found in diabetes mellitus The skin lesions are : (1) Diabetic pruritus-this may be localised or generalised. In hyperglycaemia the itching is particularly localised to the perineal and genital regions, (2) Diabetic yellowness is due to the excess of carotene in the serum of a diabetic giving rise to the carotene colour of the skin A diabetic cannot properly convert the carotene into Vitamin A, (3) Diabetic gangrene—this occurs after some injury to the toes or fingers in a diabetic, (4) Trophic ulcer—this is an indolent, punched out and painless ulcer on the planter surface of the sole in a diabetic, (5) Necrobiosis lipoidica diabeticorum—is a sharply circumscribed reddish lesion with pigmentation at the periphery in a frank or latent diabetic Commonly seen on the legs and is found mostly amongst women, (6) Fungus infection—is very common in diabetics, particularly moniliasis gets a firm foothold in a diabetic Moniliasis vulva, intertrigo, paronychia are common.

Diagnosis · (1) Typical lesions, (2) Examination of urine for sugar, (3) Blood sugar estimation, (4) Blood Vitamin A and C estimation

Differential diagnosis (1) Trophic ulcer due to leprosy and (2) Xanthoma tuberosum

Prognosis Is fair

Treatment Prophylaxis is to cut down carbohydrate intake and avoid over weight Curative is to have local treatment for fungus, trophic ulcer and pruritus together with anti diabetic regime Sometimes insulin therapy is advocated Sovental Jelly (Knoll) locally helps in pruritus

XANTHOMA

Definition Is a skin disease characterized by papular yellowish plaques of various sizes all over the body

Types (1) Xanthoma palpebrum or Xanthelasma, (2) Xanthoma tuberosum (3) Xanthoma disseminatum and (4) Xanthoma diabeticorum

Etiology Xanthometosis is due to the disturbance in the lipoid metabolism in which the liver is also concerned Hyperlipemia is associated with Xanthomatosis cutis Vitamins are also responsible for its causation

People of any age may be affected but Xanthoma palpebrum is usually found after the age of 40 Both sexes may be affected

Signs and symptoms Xanthoma palpebrum is the most common type and is found on the medial and often upper or lower eye lid of one or both eyes and is also kown as Xanthelasma (Fig No 196) The lesions are oval yellowish plaques of about ¼ to ½ inch in length The narrow end is towards the inner or outer canthus of the eyes It does not itch It is only a cosmetic dis

figurement. Xanthoma tuberosum may be a half pea-sized papule or plaque of 1 inch or 2 inches square or may be nodular in shape. Lesions are yellowish in colour. Commonly found on the joints and may be on the plams and soles. The lesions of Xanthoma disseminatus



Fig No. 196
Xanthelasma

are found on the flexure surfaces and in the axillae and mucous membranes. There is no itching. The lesions of Xanthoma diabeticorum are small yellowish papules half-pea in size and are found on the extensor surfaces associated with diabetes. Itching is present.

Diagnosis: (1) Yellow papules on the skin, (2) Blood sugar, blood cholesterol and Vitamin A are high, (3) Blood pressure is high, (4) Biopsy-histopathology shows typical Touton giant cells in the dermis. Later on fibrosis replaces the foamy cells.

Differential diagnosis: Necrobiosis lipoidica diabeticorum.

Prognosis: Good in Xanthoma palpebrum but grave in other types.

Treatment: Diet should be fat-free. Insulin is valuable in Xanthoma diabeticorum. Heparin in dose of

25 000 units twice a week for 2 weeks is also advocated Plastic surgery, improving liver function and Vitamin B_{12} therapy are advocated in Xanthelasma

PSEUDOXANTHOMA ELASTICUM

This is characterized by yellow papules on the flexure surfaces with cutis hyperplastica and without hyper-cholesterolaemia Found in axillæ and sides of the neck Histopathology shows degeneration of the elastic tissues in the dermis No treatment is known

NECROBIOSIS LIPOIDICA DIABETICORUM

Definition Is a skin disease characterized by multiple oval yellowish plaques on the extremeties

Etiology Diabetes is associated but sometimes it may develop in a non diabetic also Disturbances of cholesterol metabolism is said to be responsible Affects all

Sings and symptoms Lesion starts after some trauma and are oval, yellowish plaques of the size of the palm. Affects legs thighs and forearms and is non itching

Diagnosis (1) Typical lesion with pigmented periphery and yellowish centre, (2) History of trauma, (3) History of diabetes, (4) Blood sugar, blood cholesteral and blood vitamin A are high, (5) Histo pathology shows homogenization of the collagen fibers in the dermis with necrobiotic change Blood vessels are obliterated

Prognosis Good

Treatment Insulin injectioned, X'ray therapy, fat free and low carbohydrate diet is given

ACANTHOSIS NIGRICANS

Definition Is a skin disease characterized by pigmented papules in the axillae, groin, face and eyelids

Etiology Sometimes seen in tropical countries Is associated in elderly patients with malignant disease of the viscera which is the adult type or *malignant type* whereas in the younger people intestinal tuberculosis followed by direct invasion of the adrenals may be the cause which is the Juvenile type or *benign type* Theories for the causation of acanthosis nigricans are (1) Malignant disease of the gastrointestinal system producing in patients an altered response to light, (2) Involvement of the adrenal gland or the sympathetic nervous system, (3) Disturbance of the endocrine glands and (4) Vitamin C deficiency with hypervitaminosis A

Signs and symptoms Half pea sized black pigmented soft nodules may be found in the axillae, groins, perineum, genitalia and on the face May be found on the mucous membrane such as tongue and vagina Skin may be ichthyotic Alopecia may be associated Nail changes may be present

Diagnosis (1) Soft, pigmented nodules situated in the axillae, groin, face, (2) Ichthotic skin, alopecia with nail changes, (3) Biopsy—histopathology of the nodule shows hyperkeratosis with acanthosis and the stratum basalis shows excessive melanin formation.

Differential diagnosis Keratosis follicularis, Ichthyosis

Prognosis Adult type is grave

Treatment No treatment is known In the juvenile type thyroid extract gr ¼ daily with high Vitamin A and in the adult type injection of suprarenal extract ACTH injection in both the types may be tried

CHAPTER XVI

DERMATOSIS DUE TO AVITAMINOSIS

"Intelligent treatment by diet is the greatest weapon available to preventive medicine" The greatest achievements in our knowledge is the research in the field of protein and the synthesis of vitamins

With the progress of civilization and to adjust with the changing customs of different races there has been a revolution in the diets throughout the ages The availability or dearth of food in the tropics has a tremandous effect on the nutrition The essential elements in the body metabolism are the proteins, minerals and vitamins With the discovery of vitamins and the establishment of its correlation with diseases it has been possible now to treat various skin diseases only with vitamin therapy It has been recognised today that the vitamins are assential to maintain health and to act as a prophylaxis against various diseases

VITAMIN-A

Etiology Due to unbalanced diet avitaminosis A is quite common in the tropics Vitamin A is formed in the gut wall and is stored in the liver Deficiency may be due to (1) deficient or unbalanced diet (2) to inefficient absorption of provitamin from the intestinal tract, (3) mineral oil intake (4) deficiency of vitamin E intake, (5) infectious diseases, (6) parasites in the small intestines adversely affect the metabolism of vitamin A, (7) choline is essential for storage of vitamin A, (8) hormones

are also responsible Age—all ages are affected, Sex—both sexes are affected equally in the tropics

Signs and symptoms When the deficiency is very mild the skin is dry and on scratching fine exfoliation occurs which looks like a white line such a mild condition is called *Xeroderma* which is more aggravated in winter When the deficiency is moderate and chronic the skin becomes dry, black and cracks appear on the skin like fish scale This condition is called *Ichthyosis* (Fig No 10 & 11) when localised hypertrophy or warty growths appear on the ichthyotic skin it is called *Ichthyosis hystrix* When the condition is much advanced and the patient dies soon after birth is called *Herlequin baby*. Where the skin is like a crocodile and breathing is not possible There may be hyperkeratosis of palm and sole which is called *Hyperkeratosis plantaris et palmaris* When the vitamin A deficiency is chronic there is also besides dryness of the skin keratotic plugs appearing at the mouths of the hair follicles, sometimes with the loss of hairs, which are felt like thorns when it is called *keratosis follicularis* Skin looks stippled and gets exaggerated during winter Traumatic areas are particularly affected such as round the elbows, buttocks knees extensor surfaces of the extremities and sometimes on the body When this condition is further exaggerated the lesions conglomerate together and affect the face neck, chest, back, extremities and the skin becomes pigmented This condition is known as *Darrier's disease* Sometimes from keratosis follicularis stage the skin lesions become nodular, half pea sized in shape and get distributed round the elbows, buttocks and knees This condition is called *Phrynoderma* When the vitamin A

deficiency becomes very chronic the patient develops red pin head sized papular lesions on the skin in patches on the sides of neck, trunk, extremities, back of fingers alternating with normal skin accompanied with edema of face, body, hyperkeratosis plantaris and palmaris, there is also scaliness on the scalp with alopecia with dis coloured and cracked nails The skin lesions are symmetrical in distribution After several months the skin of the whole body becomes yellowish red in colour and somewhat atrophic Itching is the only symptom This condition is known as *Pityriasis rubra pilaris* Together with the skin lesion there may be Bitot's spots in the eyes, photophobia and xerophthalmia

Diagnosis (1) Typical dry skin alone in xeroderma and with fish scale like lesions or warty lesions in ichthyosis with pigmentation and follicular hyperkeratosis in keratosis follicularis and Darrier's disease, half pea sized nodules in phrynoderma, with patchy, red papular lesions alternating with normal skin accom panied with hyperkeratosis palm and sole in pityriasis rubra pilaris, (2) Blood Vitamin A and C estimations show low blood levels, (3) Biopsy—histopathology shows in ichthyosis there is atrophy of the sweat and sebaceous glands with thinning of the epidermis, in keratosis follicu laris keratotic plug in the mouth of the hair follicle with hyperkeratosis of the epidermis and in Darrier's disease in addition there are *corps ronds* in the stratum granulosum and increased pigmentation in the stratum basalis In pityriasis rubra pilaris there is hyperkeratosis and parakeratosis of the stratum corneum, keratotic plugs in the mouths of the hair follicles, degeneration of the stratum basalis and perifollicular infiltration in dermis.

Differential diagnosis (1) Lichen planus (2) Exfoliative dermatitis and (3) Acne vulgaris

Prognosis Good

Treatment Prophylactic dose of vitamin A is 6000 i u *daily* *A balanced diet should* consist of meat, fish, egg, vegetables, milk, butter and ghee

Curative is to give by mouth Vitamin A in dose of 100,000 i u. daily for *9 months* Sometimes multiple deficiency is present and multivitamin is advocated (Panlyn of Calcutta Chemical) Protein diet is helpful Locally lotiocalamine or Sovental Jelly (Knoll) are helpful

VITAMIN B COMPLEX

Vitamin B deficiency causes *edema of the skin* which is called beri beri

Treated with high dose of vitamin B parenterally and with protein diet

Vitamin B_2 deficiency causes ariboflavinosis Signs and symptoms angular stomatitis, inflammation of both the lips called cheilosis, dermatitis in the nasolabial folds called dyssebacia with squamous lesions over the whole of the face Tongue has typical magenta colour with atrophy of papillae There is often photophobia and conjunctivitis The genitalia presents an erythematosquamous lesion which is very itchy All these lesions together go to form the *oro genital syndrome* There may be erythemato papular lesions on the nose with ulceration of the cornea.

Treatment Daily requirement is 3 mg per day for an adult Riboflavine by injection or by *mouth is*

indicated High protein diet is helpful particularly liver, egg milk Sovental jelly (knoll) locally helps

NIACINE

Nicotinic acid amide (niacine) deficiency causes Pellagra

Definition Pellagra is characterized by erythemato squamous skin lesions with gastrointestinal troubles and irritability

Etiology This condition develops in the tropics in adults mostly but children are no exception Both the sexes are equally affected

Niacine deficiency may be due to (1) Unbalanced diet, (2) Inhibition of synthesis in the gut caused during sulpha antibiotic therapy, (3) Intestinal infection such as giardiasis amaebiasis, blantidiasis and ascariasis

Signs and symptoms The disease has a prodromal stage characterized by dyspepsia insomnia and erythematous lesions on the neck hands and legs for 2 to 3 years Gradually the early erythematous skin lesions change to dry erythemato bullous further changing to papulo squamous with pigmentation (Fig No 197) Casal s necklace is the erythematous lesion round the neck becoming pigmented in a pellagrin The skin lesions are symmetr cally distributed on the dorsum of both hands (Fig No 198) and to a variable length over the forearms dorsum of feet and the legs The face, neck and the part of the back become erythematous changing to blackish colour in the tropics The skin lesions on the face occurs on both the malor regions and on the nose like butterfly Mucous membranes look bright



Fig. No. 197
Pellagra

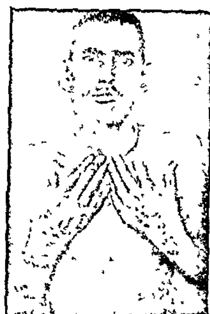


Fig. No. 198
Pellagra

red in colour. Salivation is profuse Muscular weakness develops and burning sensation is felt in the mouth. Sometimes ulceration in the angles of the mouth may be found. In the late stage the skin becomes atrophic and pigmented. The patient may have diarrhoea or constipation but sometimes alternate constipation and diarrhoea may be seen. Dyspepsia becomes chronic. Sprue or para-sprue like symptoms may be found. Insomnia, irritability, neurasthenia are commonly seen. The patient sometimes develops psychosis. Typical cases are rare. Subclinical cases of pellagra are common in the tropics.

Diagnosis : (1) Symmetrically distributed, erythemato-squamous or papulo-pigmente

dorsum of the hands, feet and round the neck with dyspepsia and irritability with insomnia in a patient, (2) Increased porphyrin in urine (3) Biopsy-histopathology shows hyperkeratosis with parakeratosis in the stratum corneum acanthosis of the stratum mucosum, demarcation of the stratum basalis with increased formation of melanin In the dermis there is edema and dilatation of vessels

Differential diagnosis (1) Eczema, (2) Leprosy, (3) Syphilitic cutis and (4) Dermal leishmaniasis

Prognosis Is good in mild but grave in acute cases

Treatment The whole vitamin B complex is given daily by injection and Niacin orally 100 mg Crude liver extract is injected intramuscularly 2c c every day. The diet should consist of milk, protein (meat, fish and egg) vegetables (tomato, peas) and fruits (mango, orange) and as a supplement multivitamin tablets Locally liniment Calamine is applied several times a day Exposure to sunlight is avoided

PINK DISEASE

Definition Is a skin disease of children characterised by redness of hands and feet with photophobia

Etiology Generally affects children from the age of three months Cause is not known Mercurial poisoning or vitamin B complex deficiency may be responsible

Signs and symptoms The child becomes restless, irritable and sleepless Anorexia is present with salivation Photophobia is marked. After about a month the hands and feet become red and cold Itching is promi



Fig. No. 197
Pellagra



Fig. No. 198
Pellagra

red in colour. Salivation is profuse Muscular weakness develops and burning sensation is felt in the mouth. Sometimes ulceration in the angles of the mouth may be found. In the late stage the skin becomes atrophic and pigmented. The patient may have diarrhoea or constipation but sometimes alternate constipation and diarrhoea may be seen. Dyspepsia becomes chronic. Sprue or para-sprue like symptoms may be found. Insomnia, irritability, neurasthenia are commonly seen. The patient sometimes develops psychosis. Typical cases are rare. Subclinical cases of pellagra are common in the tropics.

Diagnosis: (1) Symmetrically distributed, erythemato-squamous or papulo-pigmented skin



Fig No 199
Kwashiorkor
(Case Dr D B Jelliffe)

Diagnosis (1) Weaning age, (2) Diarrhoea, (3) Irritability and (4) Skin rash

Prognosis Good when treated

Treatment Prophylaxis consists of good antinatal care of mother If the mother is nursing the child as is the common custom in the tropics the diet during lactation of the mother should be scientifically balanced

Curative consists in giving repeated blood transfusion to the child orally vitamin A in dose of (10 000 i u) in divided doses with Multivitamin solution by mouth Locally Liniment calamine should be applied 8 to 10 times during the day and night When the lesions are dry 1 p c Ung Ichthyol is applied

Diet is very important The child should be given normal diet consisting of rice, bread dal (pulses), vegetable soup, meat and liver soup, fish, egg, and milk

nent which causes secondary infection. Sometimes an erythemato-papular rash is found on the body.

Diagnosis : Restless, irritable child with excessive salivation and red hands and feet.

Differential diagnosis : Eczema, Congenital syphilis.

Prognosis : Takes several months to get well. Death sometimes takes place due to intercurrent diseases.

Treatment : Sedative is given such as syrup chloral hydrate 3 to 4 times a day for several days. Protein deit. Orally vitamin-B complex and locally Sovental jelly.

KWASHIORKOR

Definition : Kwashiorkor is a nutritional skin disease of children charactrized by edema of hands and feet with pigmentation, depigmentation, hepatomegaly, *diarrhoea and mental changes with stunted growth.*

Etiology : Is not very common in the tropics as breast feeding is continued for about 2 years. Found in India. Age—below one year. Sex—both sexes suffer. It is a condition of malignant malneutrition.

Signs and symptoms : Edema is the earliest sign which appears on hands and feet. Hairs are fine, rough and look reddish. Macular errythematous rash appears on buttocks, perineum, groins, hands and fect and exfoliation with raw, red areas are often seen in infants with kwoshiorkar (Fig. No. 199). Pigmentation and hypopigmentation, microcytic anaemia, enlarged liver and associated *with palpable spleen. Diarrhoea is* always an associated early symptom. Children become fretful, restless and irritable. Growth becomes stunted.

CHAPTER—XVII

PIGMENT ANOMALY OF THE SKIN

May be (1) Hyperpigmentation and (2) Hypopigmentation

Causes of hyperpigmentation are —

A Congenital such as (1) Tropical races (2) Freckles, (3) Xeroderma pigmentosum (Fig No 161) and (4) Incontinentia pigmenti

B Physiological as in pregnancy

C Physical agents Burns

D Chemical agents Drugs and Chemicals

E Infection (1) Pediculosis, (2) Fungus, (3) Syphilis (4) Leprosy (5) Kala-Azar, (6) Malaria

F Endocrine (1) Pregnancy (2) Addison s disease and (3) Hyperthyroidism

G Nutritional (1) Pellagra (2) Melanoderma due to difficiency of Vitamin A and Vitamin C and excess of Vitamin D and (3) Malnutrition

H Dermatosis such as (a) Psoriasis (b) Lichen planus (c) Pemphigus, (d) Senile Keratosis (e) Acanthosis nigricans and (f) Melano carcinoma

I Unknown cause such as chloasma

Causes of hypopigmentation are (1) Albinism and (2) Leucoderma

INCONTINENTIA PIGMENT

Definition It is a hereditary pigmentary disease occuring in children sometimes after birth

VITAMIN-C DEFICIENCY

Causes scurvy. Clinical scurvy in the tropics is rare due to the consumption of plenty of green vegetables but subclinical cases may be seen. The scurvy is characterized by the loss of weight, bleeding from the gums and in the skin. The skin lesions consist of petechial haemorrhage with perifollicular congestion. Hematomas may also be found. Skin becomes rough.

Treatment consists of (1) rest in bed, (2) Vitamin C (500 mg.) is injected intramuscularly twice daily for a week and then vitamin C (200 mg.) to be taken by mouth every 6 hours for a week, then 100 mg. 50 mg., 25 mg. before *stopping the vitamin C therapy*. Diet should consist of milk, meat, fish, liver, vegetables, fruits (orange, lime).

CHAPTER—XVII

PIGMENT ANOMALY OF THE SKIN

May be (1) Hyperpigmentation and (2) Hypopigmentation

Causes of hyperpigmentation are —

A Congenital such as (1) Tropical races, (2) Freckles, (3) Xeroderma pigmentosum (Fig No 161) and (4) Incontinentia pigmenti

B Physiological as in pregnancy

C Physical agents Burns

D Chemical agents Drugs and Chemicals,

E Infection (1) Pediculosis, (2) Fungus, (3) Syphilis, (4) Leprosy (5) Kala-Azar, (6) Malaria

F Endocrine (1) Pregnancy, (2) Addison's disease, and (3) Hyperthyroidism

G Nutritional (1) Pellagra (2) Melanoderma due to difficiency of Vitamin A and Vitamin C and excess of Vitamin D and (3) Malnutrition

H Dermatosis such as (a) Psoriasis, (b) Lichen planus, (c) Pemphigus, (d) Senile Keratosis, (e) Acanthosis nigricans and (f) Melano carcinoma

I Unknown cause such as chloasma

Causes of hypopigmentation are (1) Albinism and (2) Leucoderma

INCONTINENTIA PIGMENT.

Definition It is a hereditary pigmentary disease occuring in children sometimes after birth.

Etiology : It is a rare disease in the tropics. It is said to be common in girls. Starts early in infancy. This is also known as Bloch-Sulzberger syndrome. Cause is not known but may be due to intrauterine virus infection or hypervitaminosis A with hypovitaminosis C.

Signs and symptoms : The child is born with hydrocephalus or with other defects. After a week from the birth the child usually develops an erythematous rash all over the body. Sometimes the rash may be bullous. The rash disappears in about a week's time leaving pigmentation all over the body. This pigmentation may start anytime from birth upto the age of two years. Pigmentation usually disappears near about puberty but often it is permanent. The child will have some malformations either in the form of alopecia, thickening of palm and sole and hydrocephalus. In a grown up child malformation of teeth or absence of teeth, occular defects, congenital heart disease, epilepsy and even paralysis may be associated. The pigmentation is found from the head *to the feet and is typical. Macular pigmentation of* various patterns are seen. Typical lesions are bizarre *arborization in distribution. The usual colour* is the slaty colour but shades of brown to black may be found. It is not itchy. Sometimes patients feel discomfort in summer due to the poor function of the sweat glands. General health may be good

Diagnosis : . (1) *Typical bizarre pigmentation of* chocolate colour of the skin of trunk in a child since birth, (2) Biopsy-histopathology shows thinning of the epidermis with hyperkeratosis and acanthosis. Large amount of melanin in the dermis and specially perivascular in distribution. Pigment granules look dropped

in the dermis and may be found in chromatophores and is called pigment incontinence

Differential diagnosis (1) Drug rash, (2) Naevus, (3) Angioma serpiginosum and (4) Melanoderma.

Prognosis So far the skin pigmentation is concerned it is usually permanent Rarely the pigmentation fades

Treatment No treatment is of any use I M injections of Vitamin C for a long time together with cortisone therapy and a diet deficient in vitamin A is helpful in some

CHLOASMA

Definition Is a localized symmetrical hyperpigmentation on either side of the face of females

Etiology No cause is known but ovarian dysfunction or liver dysfunction are found Commonly seen in middle aged women in the tropics Varieties are (1) Symptomatic when associated with uterine troubles and (2) Secondary when due to pressure or irritation

Signs and symptoms Macular pigmented spot of the size of a small coin appears on the malor region of one or both sides of the face Gradually they enlarge in size and become almost circular with about an inch in diameter Sometimes a linear pigmented patch appears on the bridge of the nose It is nonitchy and noninflammatory Colour may be brownish or blackish.

Differential diagnosis From fixed drug rash

Prognosis It is difficult to cure

Treatment Vitamin C (Redoxon 500 mg) may be injected with vitamin A (Arovit 100 000 i u) by mouth.

Testosterone propionate therapy is sometimes helpful it is due to hypersecretion of folliculin.

Locally (1) Banoquine may be applied and the fresh solution of 5 p.c. monobenzyl ether of hydroquinone should be used for 2 weeks locally. A bleaching cream is advocated containing bismuth oxychloride.

MELANODERMA

Definition : Is a chronic skin disease characterized by macular bluish or blackish ,pigmented plaques all over the body.

Etiology : It is qnite common in the tropics. Found generally in adults who do not take balanced diet. Common in adults of both sexes. Intestinal infection is commonly associated.

Signs and symptoms : Starts as multiple perifollicular pigmented macular lesions which enlarge and coalesce to form large plaques. The colour deepens and from bluish becomes black. Extremities are usually first affected symmetrically then it appears on the trunk, face and even on the scalp. The face may sometimes be affected first. It is slightly itchy. Patient is healthy and does his or her work normally. Insomnia is sometimes complained of.

Diagnosis : (1) Macular pigmented plaques all over the body in adults, (2) Estimation of blood vitamin A shows much low value, (3) Vitamin C estimation also shows a low value, (4) Gastric analysis shows hypochlorhydria, (5) Biopsy—histopathology shows slight acanthosis with excessive deposition of melanin pigments in the dermis below the stratum basalis.

Prognosis Is good

Treatment (1) Vitamin C (500 mg) is injected, (2) Multivitamin is given by mouth, (3) Locally Liniment Calamine (4) High protein diet helps

ALBINISM

Definition It is a skin disease with complete or incomplete absence of pigments in skin, hairs and eyes

Etiology It is also known as congenital leucoderma Cause is not known but there is congenital absence of pigment in the skin hairs and eyes The condition sometimes is familial Common in females Age—since birth In the tropics albinos are not rare

Signs and symptoms albino (Fig No 200) when the absene of pigment is complete but partial albinism when the absence of pigments is incomplete (Fig No 201) The skin is white The hairs are thin and white Photophobia is present Skin malignancy may develop due to the effect of the actinic rays of the sun

Differential diagnosis Leucoderma

Prognosis Is never cured

Treatment No treatment is of any use Should avoid sun 10 p c Para Amino Benzoic Acid ointment is applied Para Amino Benzoic Acid may be given by mouth in dose of 25 mg 4 times daily

LEUCODERMA

Definition This is a *skin disease* characterized by acquired absence of pigmentation from areas of skin

Etiology Classification (a) Idiopathic and (b) Secondary Common in the tropics Cause is not



Fig No 200
Albinism with multiple carcinoma
(Case of Captain S N Roy)



Fig No 201
Partial albinism

known in the idiopathic type but has been found to be associated with gastro intestinal derangement such as gastritis peptic ulcer amoebiases giardiasis blanti diasis intestinal helminthiasis intestinal tuberculosis sprue para sprue and such other cond tions which hamper the absorption of nutrition from the intestine Secondary type is due to local use of substances which inhibit melanin formation in the melanophore cells such as burns or chemicals and due to some skin diseases Seen in all ages except infants and in both sexes

Signs and symptoms Macular depigmented areas may occur anywhere on the body (Fig No 202 & 203) Usually starts as pin head spots on the extremities



Fig No 202
Leucoderma
(Girl aged 7 years)



F g No 203
Leucoderma

which coalesce and form big plaques The periphery is hyperpigmented May develop on vaccination scars (Fig. No 204) Sometimes there is a red or black mole in the centre of a small oval leucodermic patch

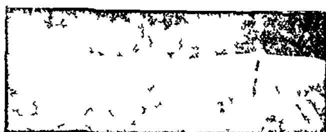


Fig No 204
Leucoderma of left forearm
(Post vaccination in a girl aged 9 years)

which is oalled *leucoderma acquisition centrifugum* Due to the absence of pigmentation the skin becomes sensitive to the sun s rays Depigmented spots may start either on the extremities or in the axillæ (Fig No 205) Itching may be present



Fig No 205
Leucoderma of axillae
(Case of Dr S C Mitra)

Diagnosis (1) Macular white plaque with pigmented periphery appearing anytime in life after infancy, (2) cause is always present either the use of rubber gloves badly tanned shoes, cosmetics, nickel watch-band or plastic watch band (3) Biposy histopathology shows absence of melanin in the stratum basalis

Differential diagonsis (1) Tinea versicolor, (2) Seborrhoea (3) Dermal Leishmaniasis (depigmented stage) and (4) Albinism

Prognosis Is fair Some get well others change colour and take a long time to develop pigmentation. There are still some which cannot be cured

Treatment Prophylaxis is to avoid exposure to sun and examine stool for intestinal infection which when found should be treated properly

Curative is to examine stool and treat the parasitic infection Then a course of emetine hydrochlor gr 1 (one grain) per day is injected intramuscularly daily for 6 days The liver is improved by methionine and choline (such as Neomethidine Neo Pharma) by injection together with Vitamin B complex by mouth Locally an ointment is rubbed containing tar but on the mucous membranes a lotion is painted containing oil of Bergomot with 60 p c Spt Rectificatus

In the tropics it is being treated by an indigenous herbal product The Indian physicians (Vaids and Hakims) have been treating leucoderma for several thousand years with the powdered seeds of Bouchi This has been put in the market as Leudermol' (Smith Stanistreet, Calcutta) ointment and oil for local use and are being used with a great amount of success Recently from Egypt another herbal preparation has claimed pigment producing function and is now available as "Meladinine" (Laboratory Grimault, Paris) lotion for application with good results.

CHAPTER XVIII

DISEASES OF HAIR

CANITIS—Is the change in colour of the hair in a young person from black to white All or few hairs may be affected Cause may be (1) Psychosomatic (2) after acute illness, (3) on patches of alopecia areata and on leucoderma, (4) deficiency of vitamin B complex and vitamin A (5) in hyperpituitarism Treatment No treatment has any effect High vitamin A (Arovit and Para Amino Benzoic Acid by mouth may be given Hair may be dyed

LEUCOTRICHIA ANNULARIS—Is a rare anomaly of the hair characterized by the presence of white bands on the shaft of the hair This is due to the presence of bubbles of air in the hair shaft

FRAGILITAS CANINUM is a condition in which the hair splits either longitudinally or transversely

TRICHORRHEXIS NODOSA is characterized by incomplete multiple transverse fractures of the shaft of tha hair which look like nodes

MONILETHRIX is characterized by multiple beads on the shaft of the hair

HYPERTRICHOSIS

Definition Is the abnormal growth of hair on different parts of the body It is also known as hirsutism

Etiology Two types are known (1) Congenital type as in nevus pilosum and in dog faced persons

where long hairs are seen all over the body including the face except on palm and sole (2) Acquired type as in pregnancy Cushing s syndrome (3) Local use of androgen (4) In women androgen produces growth of hair (5) In virilism

Signs and symptoms Partial congenital type is seen in nevus pilosus Localized hypertrichosis is commonly seen on the sacral region in case of spina bifida (Fig No 206) Excessive growth of hairs may occur on face of women Commonly seen in women



Fig No 206
Hypertrichosis Tuft of hairs on the sacral region of a child aged 2 years

after menopause It produces a psychological upset Investigation in young woman with hirsutism are (1) 17 ketosteroid high in urine (2) Skiagram of sella tarcica abnormal (3) Pyelogram (4) Abnormal carbohydrate tolerance test

Treatment Depends on the cause When due to pituitary tumour X ray therapy is helpful Operation is advised in case of suprarenal tumour Epilation

by electrolysis of the hairs is helpful. The hairs may be bleached with the application of hydrogen peroxide

HYPOTRICHOSIS

Also known as alopecia or baldness

Varieties of alopecia : (1) Idiopathic, (2) Secondary and (3) Psychic

(1) Alopecia idiopathica may be due to (a) when the hairs and hair follicles also are absent at birth together with defective dentition is known as *congenital ectodermal defect*, (b) Baldness which starts from the frontal regions and progresses backwards in young adults is called *lopecia prematura*. Alopecia generally develops in the old age due to the changes in the gonads and is known as *Alopecia senilis*

(2) Secondary alopecia may be due to (a) Hair dressing, (b) Massage of scalp, (c) Fungal, (d) due to burns, (e) due to skin diseases like scleroderma, lupus erythematosus, lupus vulgaris, syphilis (Fig No. 207).



Fig. No 207
Syphilitic alopecia

(f) due to seborrhera, (g) due to coccal infection of the hair follicles causing baldness of various shapes.

This type of baldness is called *pseudopelade de Brocq*, (h) Leprotic alopecia may occur anywhere on the body but is commonly seen on the lateral sides of the eyebrows and extremities (i) Irritation from friction of the hairs over the back of the head produces irregular shaped keloid like plaque with pustulation and alopecia which is known as *folliculitis keloidalis*

3 Alopecia due to psychoneurosis are mainly of two types (a) trichotillomania and (b) alopecia areata

Trichotillomonia occurs in insanes and hysterics The individual pulls out hairs of the scalp and produces irragular baldness Some women are in the habit of picking off hairs producing a linear alopecia Conjunctival reflex is usually absent in the patient Hair soon returns *Alopecia areata* may occur anywhere on the body Common sites are the scalp (Fig No 208 & 209), bea d moustache eyebrows and pubic region but can be



Fig No 208
Alopecia areata in a child



Fig No 209
Alopecia areata in an adult

found on any hairy region of the body Conjunctival and faucial reflexes are usually absent The lesions are circular with a diameter of one fourth to two inches

and broken exclamation mark like hairs are found at the periphery of the lesions with slight redness.

Causes are (1) Psychosomatic background, (2) Error of refraction, (3) Septic focii. The baldness is characterized by a circular patch with mild erythema. There may be one isolated patch or several. Sometimes patches may coalesce to form gyrate figures. Rarely the alopecia is universal in type. May occur at any age. Both sexes are equally affected.

Prognosis : is good but takes a long course to get well. Single or multiple patches take about a year to get well but sometimes the patches take half the time to be cured. Alopecia universalis is not possible to cure.

Treatment : There is no treatment for alopecia idiopathica. Secondary alopecia may be treated according to the cause. In seborrhoea it is advisable to treat seborrhoic capitis. When due to specific cause such as syphilis and leprosy proper treatment of these diseases when instituted alopecia improves. Pseudo-peladede Brocq has no treatment. *Folliculitis Keloidalis* with 5 p.c. ung. sulphuris. Radio-therapy is advocated. Plastic surgery may be helpful. A bromide mixture with valerian is helpful. Vitamin A (Arovit) in high dose may be given for a long time. Psychotherapy may be advised. Locally Bepathine lotion may be rubbed.

Locally 20% glacial acetic acid application may be helpful. Rarely ultra-violet light may be given locally or *Hydrocortisone* ointment (Roussell) locally. Cortisone by mouth and Vitamin B-12 in high dose may be useful.

CHAPTER XIX

DISEASES OF NAIL

When the nails are absent on fingers and toes the condition is known as *Anonychia* Supernumerary nail is associated with supernumerary finger

The nails of fingers alone may be all white or together with the nails of toes since birth in a normal indivibual when it is called *Leuconychia* This condition may be familial Sometimes "white spots' appear on the nails called "gift spots" The whiteness appears first near the lanula which comes to the free edge of the nail with the growth of the nail Sometimes only one nail is involved These white spots are due to the imperfectly keratinized cells or due to the presence of air between the epithelial cells When the nails are separated from the nail bed it is called *Onycholysis* or Onychomadesis When longitudinal splitting occurs in one or many nails as congenital or acquired condition it is called *Onychorrhexis* Due to nutritional deficiency transverse lines appear on the nail plate which is known by the name of *Beau's lines* Sometimes there is lateral growth of the toe nail which acting as a foreign body produces pain and inflammation and granulomatous tissue fomation which is called *Unguis incarnatus* or ingrowing toe nail When changes are found also in the skin, mucous membrances and hyperhidrosis of palms and soles with dystrophic, deformed and thickening of the free margin of the nails it is called *Pachyonychia congenita.* The dystrophic disturbances in the nails

when formed along with abnormality in teeth and hairs and saddleback nose producing a Mongolian facies it is called *Congenital ectodermal dysplasia*. Due to the use of ill-fitting shoes and as a result of disease like leprosy there is hypertrophy of the body of the nail as a result of which the free margin of the nail grows as an arc of a circle to about 2 to 3 inches when it is called *Onychogryphosis*. In chronic microcytic anaemia and as a result of subungual growth the shape of the nail changes like the spoon when it is called *Koilonychia*. Rarely there may be a single longitudinal groove found in one of the nails and the condition is called *Dystrophia mediana canaliformis*. Both as a hereditary condition and as a sign of some disease the finger tips may be bulbus when the condition is called clubbing of the fingers.

Pigmentation of nails is sometimes seen due to handling of medicine. Discolouration of the nail may be due (1) Syphilis, (2) Leprosy, (3) Ringworm, (4) Moniliasis, (5) Eczema, (6) Psoriasis, (7) Lichen planus, (8) Cyanosis, (9) Anæmia, (10) Nail varnish, (11) Subungual melanoma When the nail folds are also involved together with nail plate it is called *paronychia*. Those who work with water or handle chemicals and do washings usually suffer due to tinea or monilia infection. The maid and the housewife are usually victimized. Redness appears on the nail-fold then there is pus on pressure. There is tenderness also.

Treated by paintinting (1) 1 p.c. aquous gentian violet and keeping the hands and feet away from water, (2) 5 p.c. Acid Salicylic in collodion flexile. Rarely cases improve with small doses of X-ray

ONYCHOMYCOSIS

Is the infection of the nails with ringworm or monilia

Signs and symptoms Sometimes associated with ringworm or monilia infection of the skin In ringworm the nail plate is involved first and then the nail bed but in monilia the nail folds are commonly affected first and then the nail plate. The nail becomes discoloured, thickened and friable or may get hypertrophied One, two or all the nails may be affected

Diagnosis (1) Discoloured cracked, thickened and friable nails of fingers alone or both fingers and toes, (2) Skin scraping after warming with 10 p c aquous sodium hydrox sol or nail clipping with 40% sol and when examined under microscope mycilia can be seen, (3) Culture of nail clip or skin scraping in Seboroud's media will show growth of fungus (4) Biopsy histo pathology will show fungus in the layers of nail plate

Differential diagnosis (1) Psoriasis of the nail and (2) Acrodermatitis perstans

Prognosis Fair but is very intractable

Treatment Scraping the nail daily after soaking with 20 p c aquous sodium hydroxide and then applying paints containing chrysophanic acid or 5 p c Cignolin (Bayer) or Derobin (Glaxo) in acetone Surgical removal or radio-therapy may sometimes help

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